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Archives OF OPHTHALMOLOGY

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2	20	1.67	6.0
3	16.7	2.00	9.0
4	14.3	2.33	12.0
5	12.5	2.67	15.0
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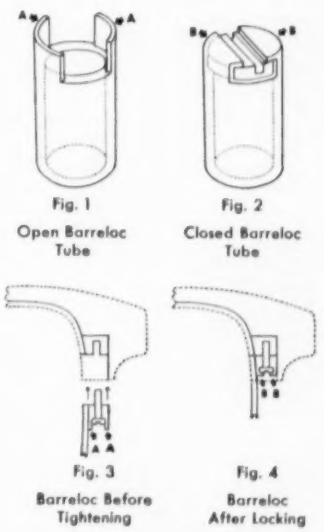

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Fig. 3 Barreloc Before Tightening

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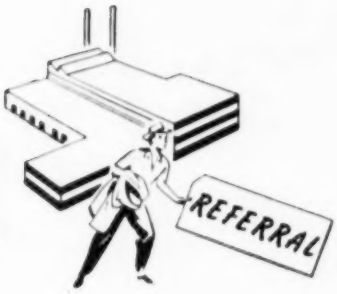

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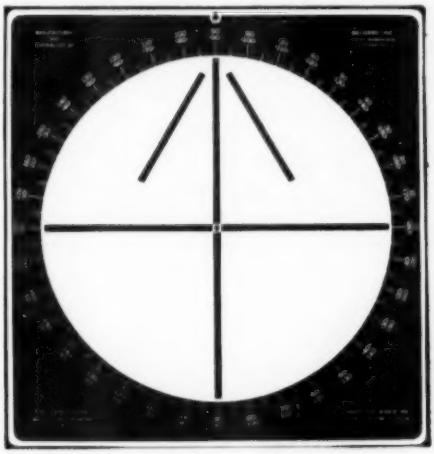

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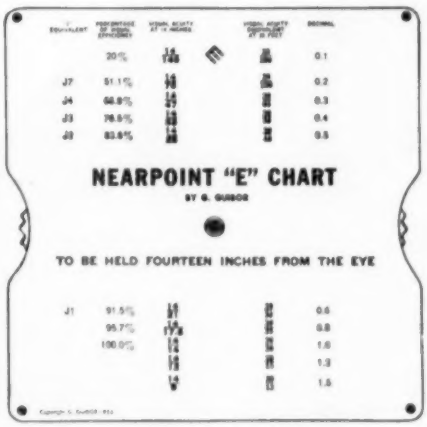

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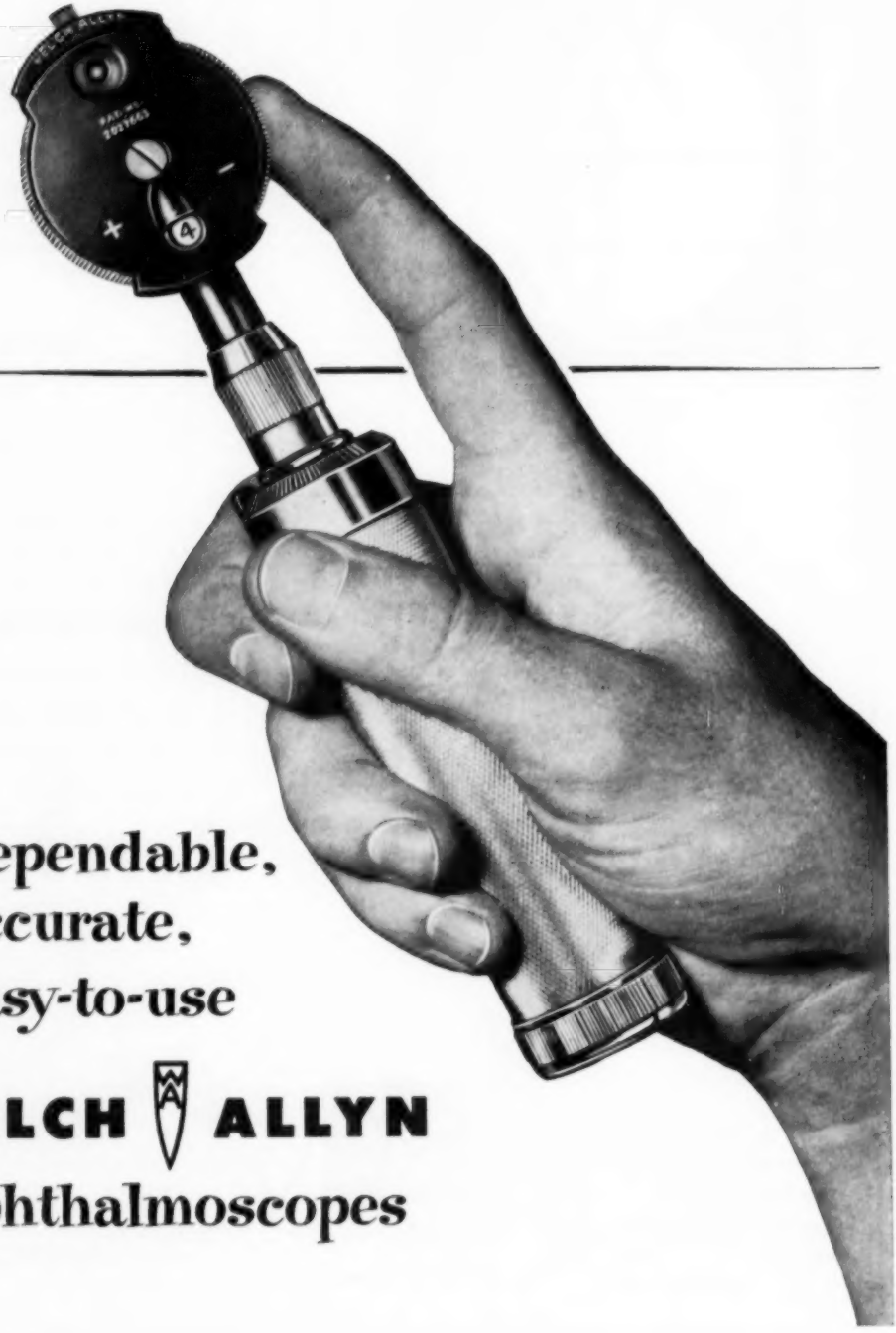

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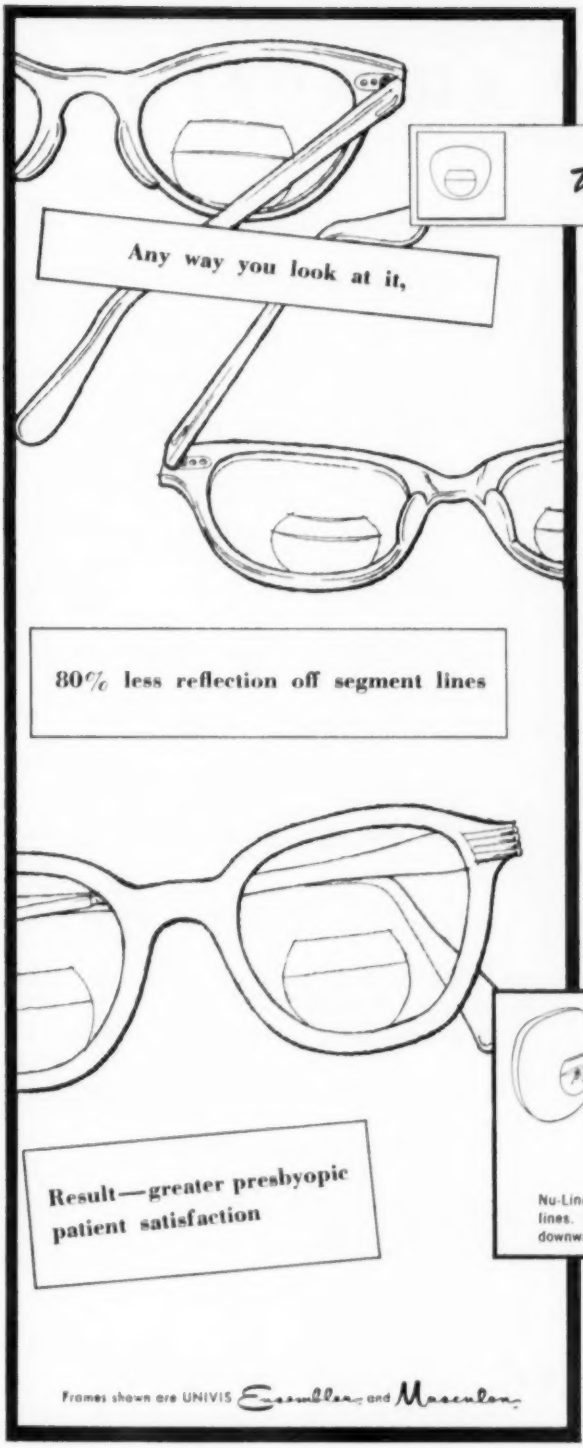
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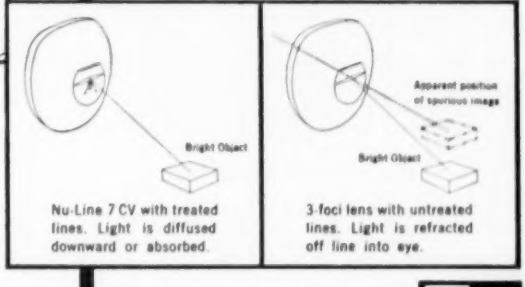
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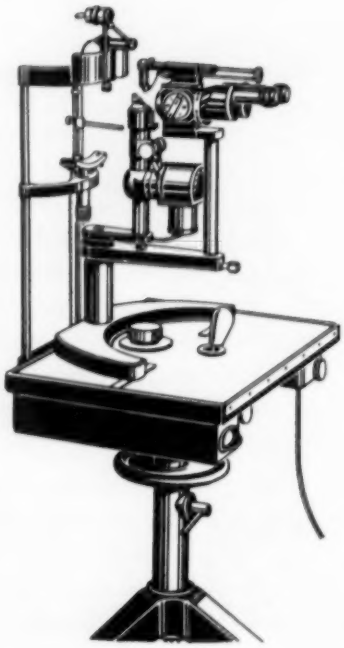

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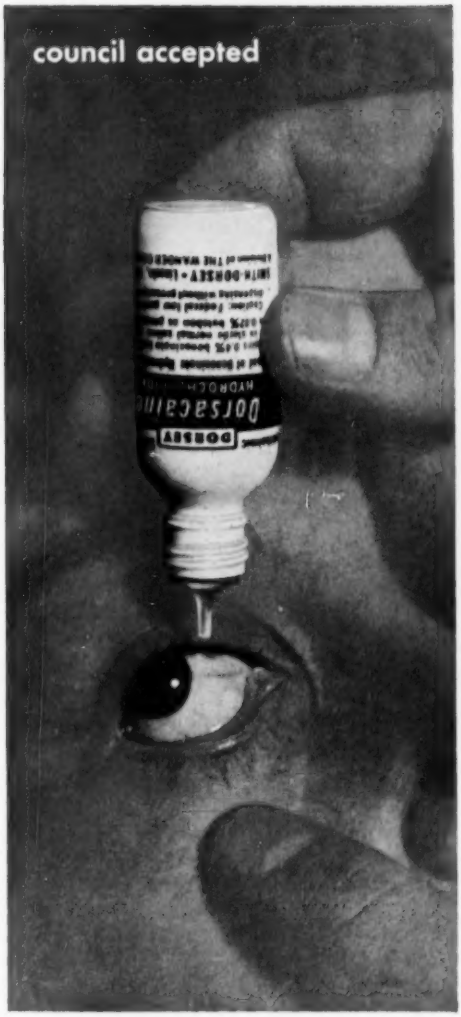

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*Schlegel, H. E., Jr., and Swan, K. C.: A. M. A. Arch. Ophth. *51*:643 (May) 1954.

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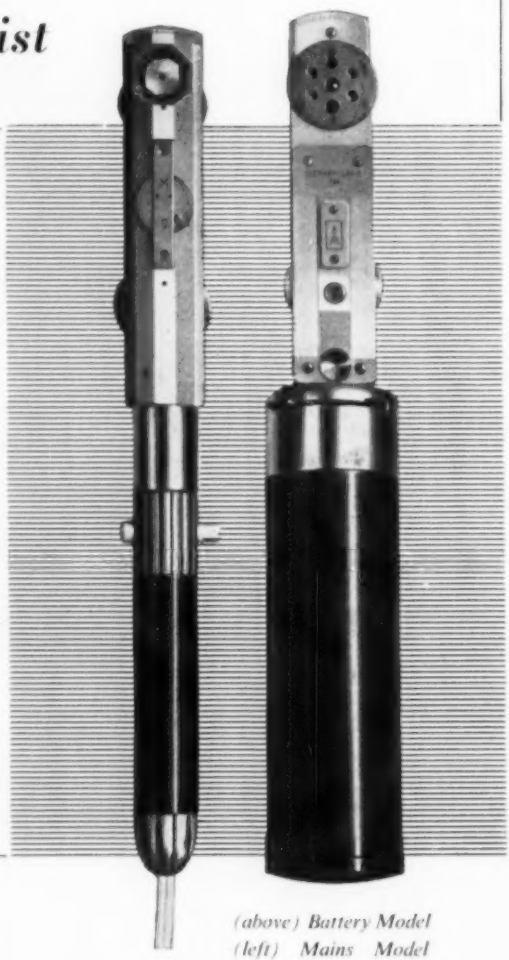

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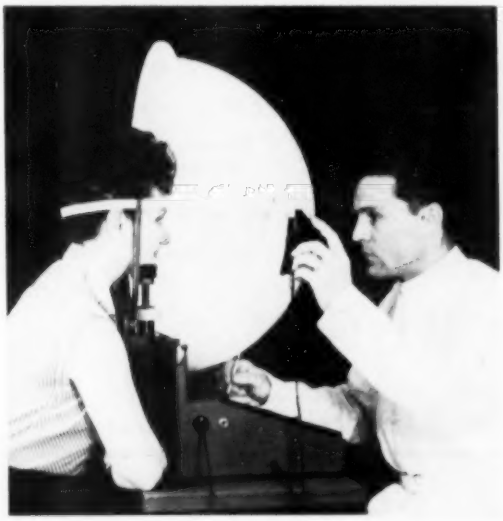

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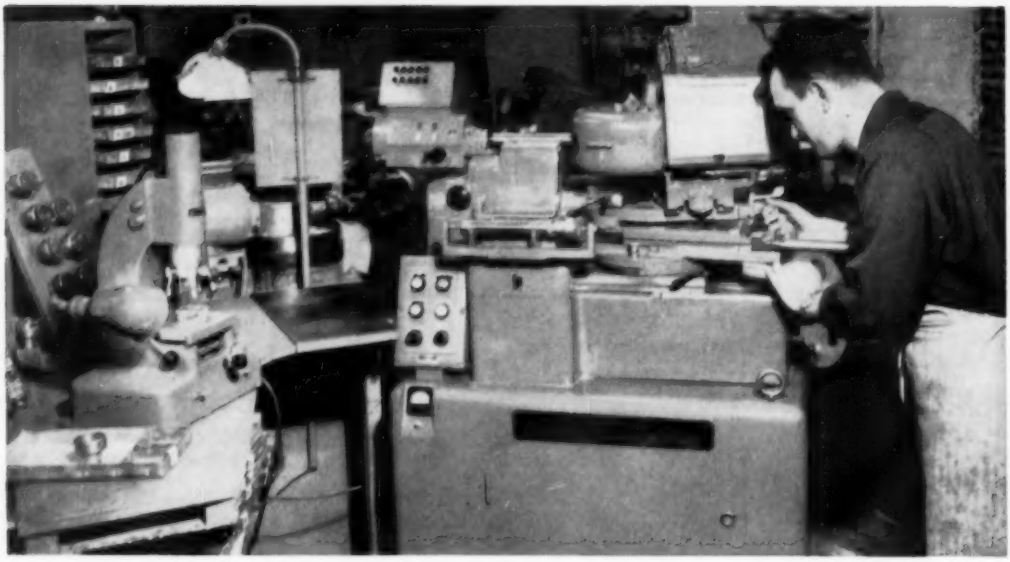

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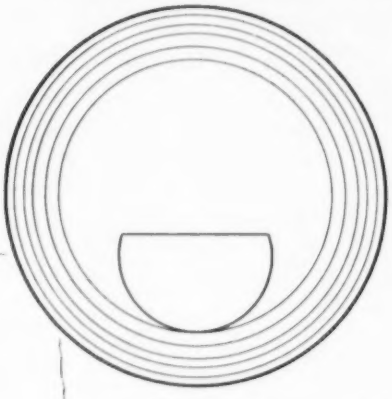

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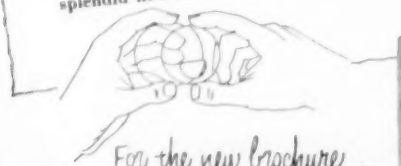

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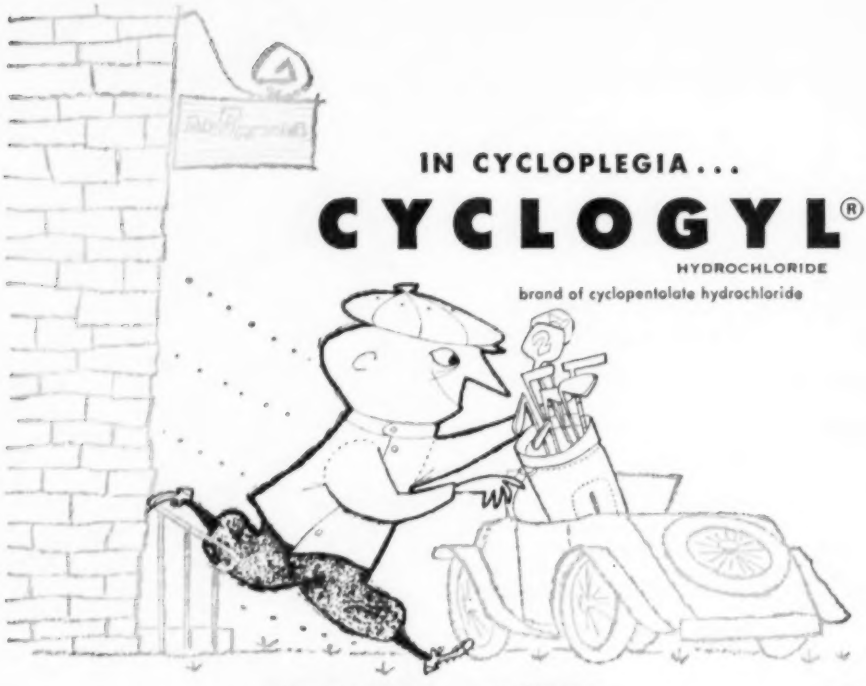

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1. Gordon, D. M., and Ehrenberg, M. H.: Am. J. Ophth. 38:831 (Dec.) 1954.

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Visibility of the Ring of Schwalbe and the Trabecular Zone

An Interpretation of the Posterior Corneal Embryotoxon and the So-Called Congenital Hyaline Membranes on the Posterior Corneal Surface

HERMANN M. BURIAN, M.D.
ALSON E. BRALEY, M.D.
and
LEE ALLEN, Iowa City

About two years ago our attention was drawn to glassy ring-like structures at the periphery of the posterior surface of the cornea. It soon became evident that these bands corresponded to the so-called posterior embryotoxon of the cornea (Embryotoxon corneae posterius, Axenfeld [1]), posterior marginal dysplasia of the cornea (Streiff [2]), congenital hyaline membranes on the posterior surface of the cornea (Mann [3]), or peripheral refractile postcorneal rim (Graves [4]). Once our interest in this subject was awakened, we devoted a close study to the limbus of all patients whom we saw and performed a slit-lamp gonioscopic examination on those who presented to external slit-lamp study an unusual appearance of the limbus or of the periphery of the cornea.

These clinical investigations were followed by a careful study of 600 pathologic specimens of eyes, and this study, in turn, prompted us to extend our investigations to the chamber angle of the eyes of pigs, dogs, rabbits, and cattle and the embryology of this region in man. Inevitably, this led us also to the problem of juvenile glaucoma.

In the present paper the results of our clinical work will be presented. The other aspects of the study will be discussed in subsequent papers * and will be mentioned here only insofar as is necessary to support our evidence.

From the Department of Ophthalmology, State University of Iowa College of Medicine.

Condensed version of a paper read at the 90th Annual Meeting of the American Ophthalmological Society, Glacier Park, Mont., June 18, 1954.

I. CLINICAL FINDINGS IN "NORMAL" EYES

Before the embryotoxon is discussed, a description of the slit-lamp appearance of the normal limbal areas will be given, insofar as it has a bearing on the subject under consideration.

When one focuses a very narrow slit beam on the sclera to the nasal or the temporal side of the globe and slowly moves the beam toward the limbus, there will suddenly appear, when the beam is near the limbus but still in nontransparent tissue, a golden glow in the region of the chamber angle which in its character is comparable to the yellowish-golden appearance noted when one is observing ocular tissues in specular reflection (Fig. 1*A*). As the beam is moved further corneaward on the limbus, one observes invariably, if the beam is carefully focused on the posterior corneal surface, a more or less translucent condensation of tissue which presents a fine, regular, or crisscross striation (Fig. 1*B*). In the majority of cases this band is perhaps 0.33 to 0.5 mm. wide; in a few it is only a small fraction of a millimeter wide, and in others it may be more than 1 mm. in width. Because of the vertical orientation of the slit beam and the width of the external scleral limbus in the upper and lower parts of the corneal circumference, this band is best observed on the nasal and temporal sides.

* References 5 and 6.

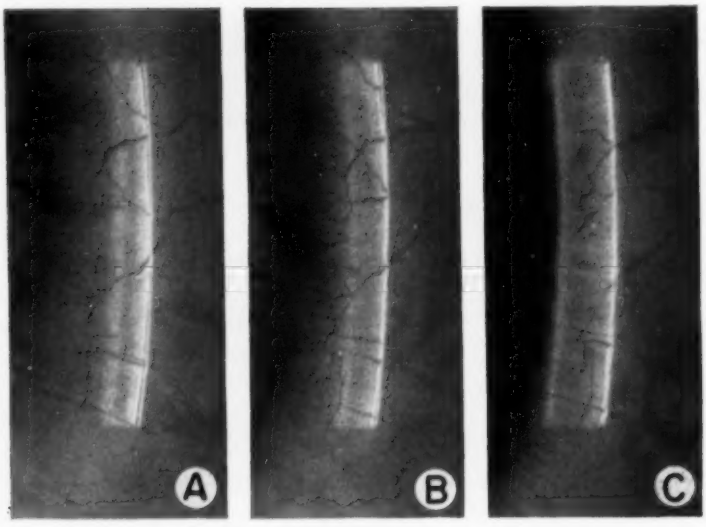
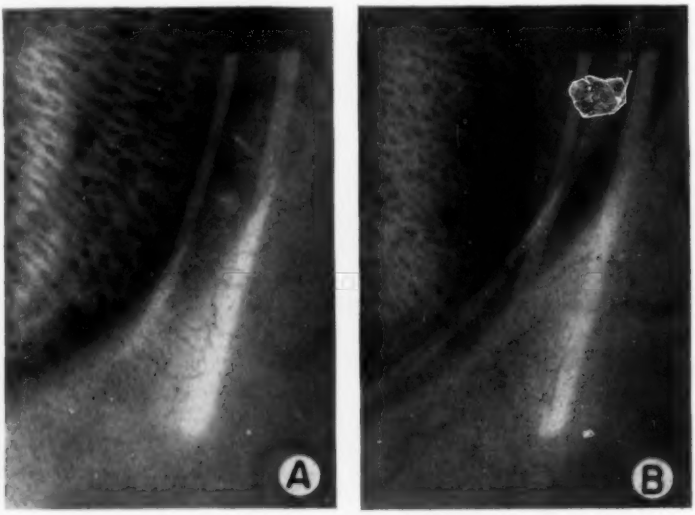


Fig. 1.—External slit-lamp appearance of average trabecular zone on temporal side.

Peripherally this band, as it appears to blend with the tissue of the scleral septum (Busacca [7]), has no discernible border, and disappears behind the wedge of the external scleral limbus, but toward its corneal side it nearly always ends in a rather well-defined edge (Fig. 1*C*). As a rule this edge is nothing more than a line delimiting the band of relucent tissue. Occasionally it was seen to consist of a distinct whitish line, concentric with the limbus, clearly thickened, sometimes slightly wavy, and in the more pronounced cases obvious in focal illumination without the aid of magnification. It must be emphasized that this line appears whitish against the darker iris only in direct focal

Fig. 2.—*A*, widely exposed trabecular zone without prominent border of Patient B. P. *B*, wide trabecular zone with prominent border of Patient R. L.



illumination. When seen in retrograde illumination, the line is always translucent and refractile.

The lower temporal portion of the left eye of Patient B. P., a 21-year-old woman, who was seen in our clinic for refraction, is pictured in Figure 2*A*. She had an unusually broad band of relucent tissue at the posterior corneal surface in the limbal area. This tissue feathered out to a thin, almost indistinguishable edge on the corneal side. It had no prominent border, except for a possible very slight indication in the upper temporal region. Just central to the edge of the relucent tissue there were fine circular lines in Descemet's membrane which appeared refractile by retroillumination but were slightly relucent in direct illumination. Figure 2*B* contrasts the appearance noted in this patient with that of a patient in whom the relucent tissue, of which there is a similarly broad expanse, is delimited by a distinct, whitish, somewhat prominent line.

This refractile line is seen in some patients only along a short stretch of the circumference, usually in the lower temporal or lower nasal periphery. It may extend along the greater part of the temporal or nasal periphery or both, and in some cases it is continuous, leaving free only the region from 11 to 1 o'clock, where we have seen it externally only in exceptional cases. Short stretches of a prominent line are frequently noted, but more extensive lines are by no means rare. After we had seen over 30 patients with more or less extensive prominent lines, the pattern was so well established that we continued using gonioscopy only on eyes in which features of special interest warranted such examination.

When a prominent line is present, observation with the slit lamp creates the definite impression that the line constitutes the edge of a "hyaline" membrane adherent to the posterior corneal surface and extending into the chamber angle. The picture agrees with that of the entity described as posterior embryotoxon of the cornea, as posterior marginal dysplasia of the cornea, or as congenital hyaline membrane on the posterior surface of the cornea.

The similarity to this clinical entity was enhanced by the frequent finding of small pigment dots on the prominent edge of the "membrane," or "opacity," and of fine strands of iris tissue inserting on it which were occasionally visible with the slit lamp.

When the translucent band was pronounced, the corneas were usually a little smaller in the horizontal diameter than normal. The condition occurs also, however, in corneas of normal size, and we have even observed it in two cases of megalocornea. Often there was also some hypoplasia of the anterior mesodermal layer of the iris. This hypoplasia was as a rule mild, but was quite evident in certain patients. Most of our patients had gray or blue irises, but there were also some with dark-brown irises, and we have seen the anomaly in two Negro patients. Most patients in our clinic are of Central European or Scandinavian stock; very few are Negroes. Studies on clinical material in which patients of Southern European stock or Negro patients are prevalent are likely to show that the anomaly occurs with equal frequency in these patients. Biozzi and Lugli [8] were able to collect 16 cases in the clinic in Bologna, Italy, in a short period.

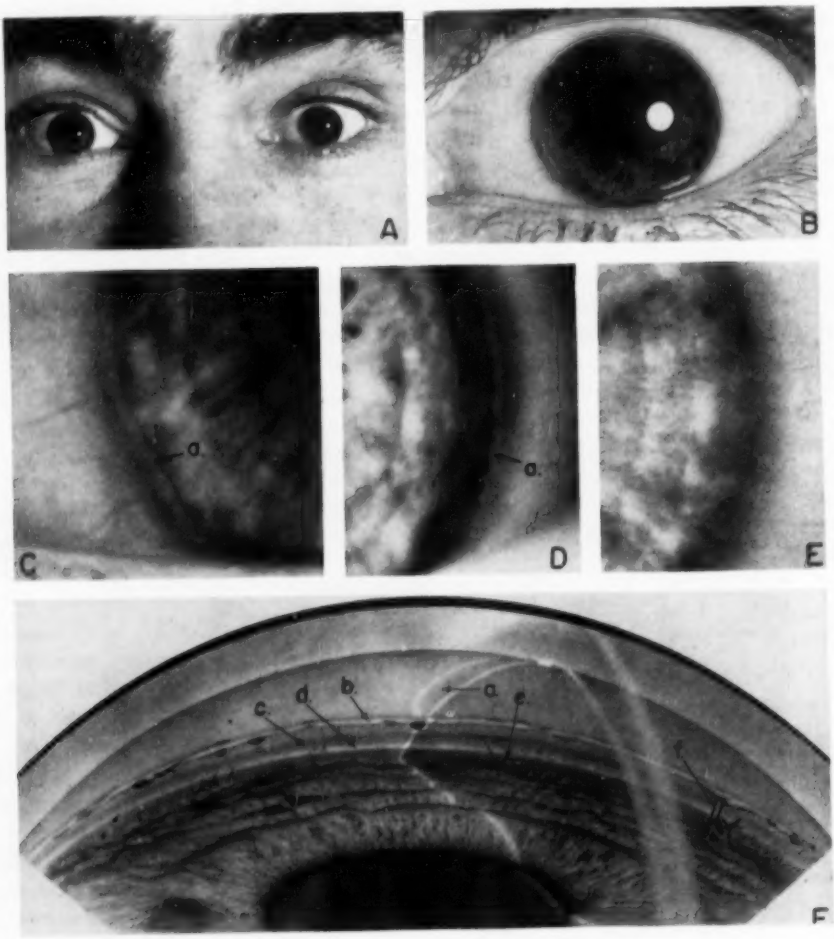
Aside from the hypoplasia of the iris, none of the eyes which we examined showed abnormal conditions which could in any way be connected with the "embryotoxon," except for a group of patients in whom an extreme development of this anomaly was found, together with iris atrophy and glaucoma. These cases will be discussed later.

The question arose, What is the anatomic substratum of the "opacity" or "membrane," and of the prominent line seen at its edge? We expected and obtained an answer to this question from gonioscopic examination.

These examinations revealed that the structures of the chamber angle were well visualized and that they had their usual appearance. There was no abnormal membrane of any kind covering the structures of the chamber angle or the periphery of the

cornea. In the patient whose slit-lamp picture is reproduced in Figure 2*A*, the trabecular region was perfectly normal for a person of her age, although it appeared slightly broader than usual, especially in certain areas. The region of Schlemm's canal showed a normal gray appearance. Ahead of this region, still within the trabecular area, was a whiter band, which blended softly into another gray region just behind the anterior edge of the trabecular zone. In this patient no prominent line was visible anywhere. In those patients, however, who presented externally a prominent line, one noted, when following the anterior border of the trabecuportion of the angle, the anterior border of lar band, that, along a greater or smaller this band became a prominent pearly-white line. In some patients clumps of pigment were seen on this line, and a certain number

Fig. 3.—Patient M. B.: *A*, photograph of patient's eyes showing cornea O. S., smaller than cornea O. D.; *B*, enlarged view of O. S., showing prominent bands nasally and temporally; *C*, lower nasal quadrant of limbus O. S. seen with direct illumination, showing prominent ring (*a*); *D*, temporal limbus O. S. with prominent ring (*a*) seen in sclerotic scatter; *E*, nasal limbus O. D., showing no prominent ring; *F*, gonioscopic view of lower angle O. S.: (*a*) corneal wedge; (*b*) prominent anterior border-ring of Schwalbe with pigment clumps; (*c*) blood-filled canal of Schlemm; (*d*) scleral spur; (*e*) ciliary body band; (*f*) bridging iris processes.



of iris processes inserted on or below the line. There were variations as to the extent and prominence of the line and as to the amount of pigment, but the basic pattern was essentially the same in every case we have observed.

From our gonioscopic findings it was evident that what we had taken to be a "hyaline membrane" in the external slit-lamp examination of these patients was nothing else than the trabecular area and that the prominent ring was the anterior border-ring of this area, or ring of Schwalbe.

We thereupon went back to an investigation of patients who did not show an "embryotoxon" and were soon convinced that the trabecular region can be visualized externally with the slit lamp in every patient at the nasal and temporal portions of the limbus. The "golden glow" at the limbus is produced by illumination of the trabecular region, and more peripherally of the scleral septum, in specular reflection, and the opacity seen in direct illumination and described at the beginning of this paper is, in fact, due to the visualization of the trabecular zone.

The findings in the numerous patients with "embryotoxon" were rather uniform, and we shall, therefore, as an example, report only one case, that of a 29-year-old male school teacher who presented himself for examination because of eyestrain and headaches. He had a mild hypermetropic astigmatic error, normal visual acuity in each eye, and normal intraocular pressure. His eyes were clinically normal except for a slight difference in the horizontal diameter of his corneas (O. D. 12 mm.; O. S. 11 mm.), a slight hypoplasia of the anterior layer of the left iris, and a prominent ring of Schwalbe in that eye. In external slit-lamp view, a few iris processes were seen to insert on that ring. The limbal area of the right eye presented no abnormality. The external and gonioscopic appearances of the eyes of this patient are shown in Figure 3.

II. CLINICAL FINDINGS IN GLAUCOMATOUS EYES

While the majority of the eyes in which we noted a prominent anterior border-ring of Schwalbe by external slit-lamp examination were clinically normal, we have observed such rings also in a number of eyes with glaucoma. The ages of these patients varied from 2½ to 83 years.

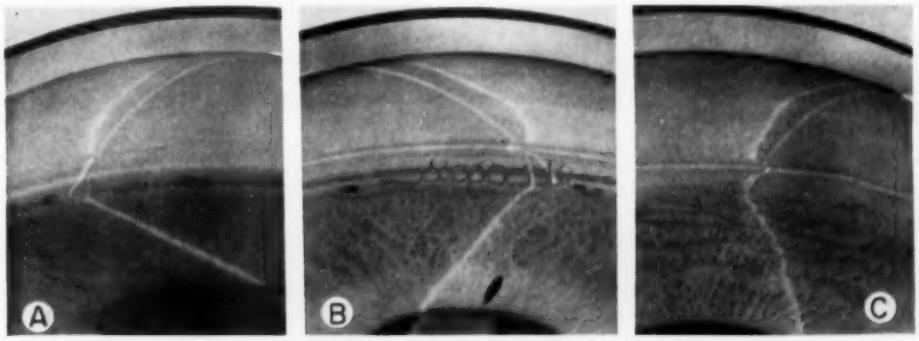


Fig. 4.—*A*, lower angle, O. D., of Patient M. K.; *B*, lower angle, O. D., of Patient J. L.; *C*, upper angle, O. D. (reversed) of Patient W. L.

In a number of instances the ring was observed as a familial occurrence. In two of these families the prominent ring was the only anomaly present. In another family only one member had a glaucoma combined with the ring; in a fourth family glaucoma and ring occurred together in a number of members.

We shall briefly report on the two families in which glaucoma occurred. In the first, we saw grandmother, daughter, and grandson. The grandmother, a 54-year-old woman, presented in both

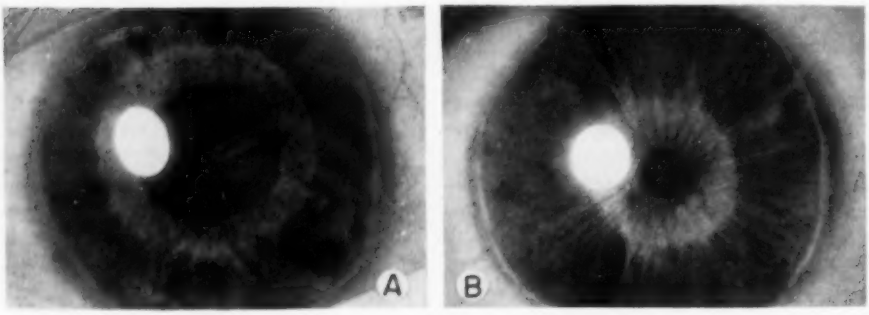
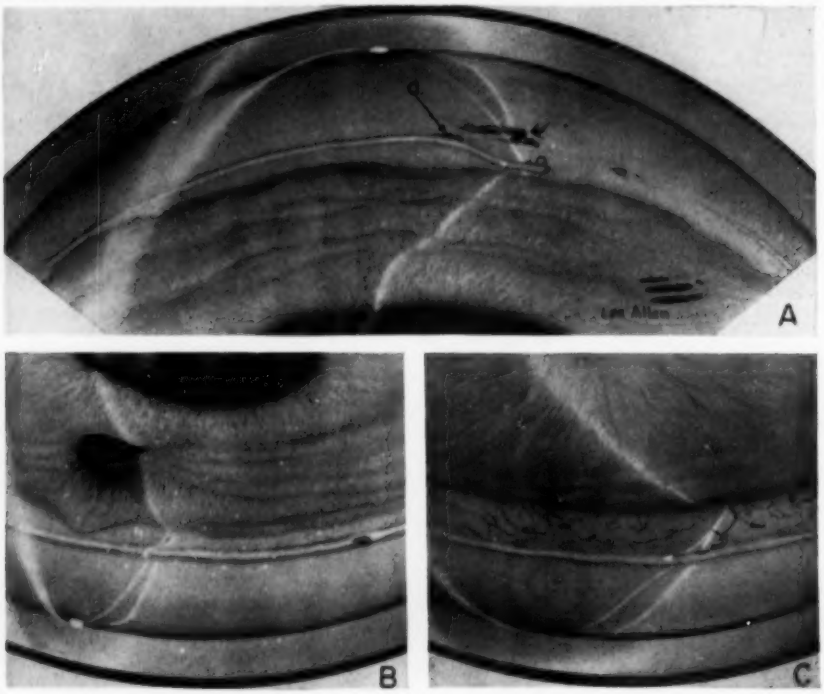


Fig. 5.—*A*, external view, O. D., of Patient J. L., showing prominent ring and marked iris atrophy with hole formation; *B*, similar view of Patient R. L.

eyes a prominently visible anterior trabecular zone and a prominent anterior border-ring, as well as marked iris atrophy. This patient had advanced chronic glaucoma. Gonioscopy revealed the angles to be open. The gonioscopic findings were essentially the same in the various regions of both chamber angles. The view of the lower angle of the right eye is illustrated in Figure 4*A*. Figure 4*B* shows the gonioscopic appearance of the right chamber angle of this patient's 30-year-old daughter, who also had a prominent ring of Schwalbe and iris atrophy. This patient had no ocular complaints, and her intraocular pressure was normal. Her 7-year-old son showed the same anomalies, and the gonioscopic view of the upper quadrant of his right eye is reproduced in Figure 4*C*.

These three patients have a number of interesting anomalies in common. All three have an iris atrophy, which in the grandmother is associated with advanced glaucoma; all three have an abnormal vessel,

Fig. 6.—*A*, gonioscopic view of lower quadrant, O. D., of Patient J. L.: (*a*) point at which ring of Schwalbe becomes detached. *B*, upper quadrant of chamber angle, O. S., of Patient J. L.; *C*, upper quadrant of chamber angle, O. D., of Patient R. L.



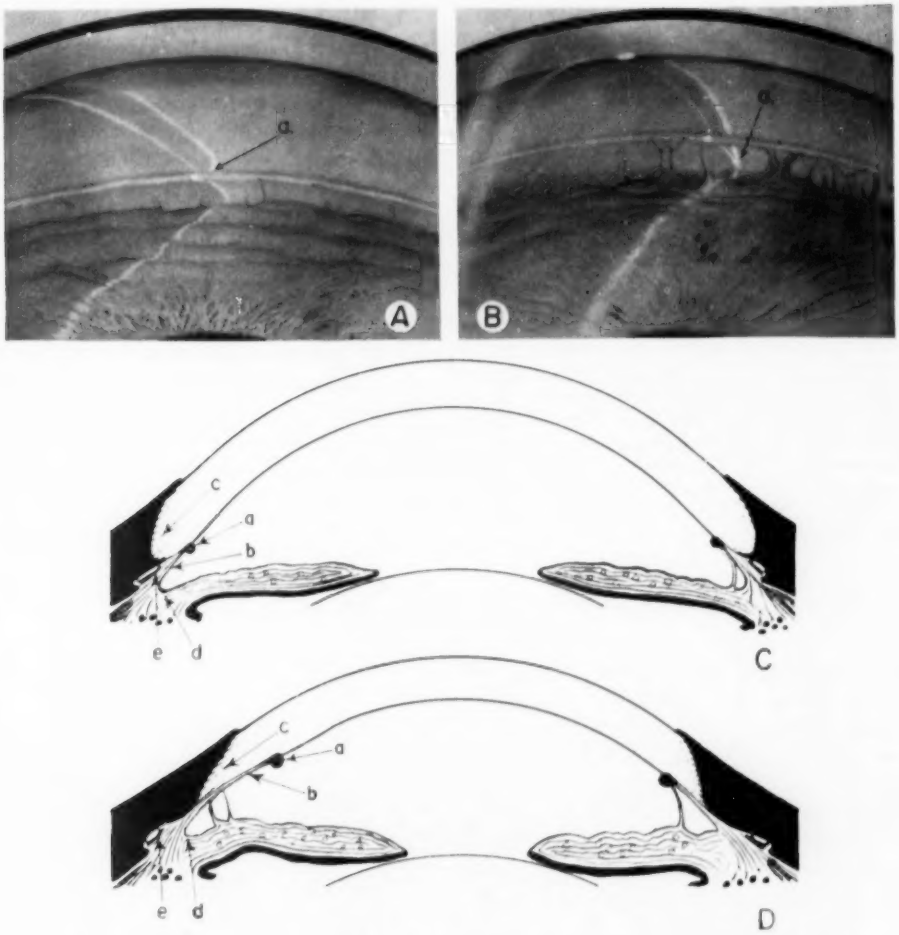


Fig. 7.—*A*, chamber angle of Patient R. K.: (*a*) end of corneal wedge. *B*, chamber angle of Patient R. L.: (*a*) end of corneal wedge. *C* and *D*, schematic interpretation of the probable cross sections of the chamber angles of these patients. *C*, Patients R. K., with cornea of relatively normal width: (*a*) normally located prominent ring of Schwalbe; (*b*) trabecular zone of normal extent; (*c*) normally shaped corneoscleral junction and external scleral limbus. *D*, Patient R. L., with a relative microcornea: (*a*) centrally located prominent ring of Schwalbe; (*b*) long, thin forward extension of uveal portion of trabeculum; (*c*) abnormal corneal wedge forming an acute angle due to peculiar distribution of scleral tissue. Note forward position of iris root (*d*) in Patient R. L. (*D*) and greater distance between apex of angle and Schlemm's canal (*e*) than in Patient R. K. (*C*).

probably an exposed major circle located at the apex of the angle, and present a line of Schwalbe of varying prominence. The grandmother and the mother show a number of coarse and fine iris processes bridging the angle of the anterior chamber.

The most advanced glaucoma and the most prominent rings were seen in two brothers, aged 35 and 38, respectively, who also had essential iris atrophy. Their 50-year-old sister and the 5-year-old son of one of them also had glaucoma and prominent rings.

The external appearance of the eyes of the two brothers is shown in Figure 5*A* and *B*. In Figure 6 gonioscopic views of their eyes are presented. The iris atrophy is clearly noted, as is the white cord-like strand in the position in which the ring of Schwalbe is expected to be found. We wish especially to call attention to Figure 8*A*, in which the

line of Schwalbe suddenly separates from the overlying corneal tissue, terminating at about the 5:30 o'clock position with a small translucent yellow ball near its free end.

Another noteworthy abnormality, seen by us also in some other patients, was found in the younger of the two brothers. The slit beam demonstrating the corneal wedge in optical section extended across what was assumed to be the trabecular region to the iris root in the lower angle, and halfway to the iris root in the upper angle. It is instructive to compare the relationship of the chamber angle structures to the corneal wedge of this patient with that of another patient. The latter, a 37-year-old man, had essentially normal eyes, but presented on external slit-lamp examination a prominent ring of Schwalbe around the entire circumference of the limbus in both eyes. The elevated, cord-like band of tissue at the anterior margin of the trabecular zone was well visualized in gonioscopy, but the corneal wedge had the usual nicely rounded form where it coincided, presumably, with the anterior border of the trabecular zone (Fig. 7).†

III. MICROSCOPIC ANATOMY OF THE CHAMBER ANGLE

We have made a careful histologic study of the region of the chamber angle in 600 eyes available to us in our collection of histologic specimens. Of most of these eyes we had at our disposal a number of sections stained with hematoxylin-eosin and with the Held-Van Gieson and Mallory stains. The result of these studies as they relate to the trabecular zone and its anterior border will now be summarized.

A. Trabecular Meshwork

The elements of the trabecular meshwork are distributed in typical fashion, and its fibers can be divided into several groups on the basis of the course of the fibers and their relationship to adjacent tissues. The fibers of the various groups differ also with regard to their constituent parts.

The following four groups of fibers can be distinguished, of which Group 1 represents the corneoscleral group, while Groups 2 to 4 belong to the uveal meshwork, according to the terminology introduced by Virchow.[10]

1. The corneoscleral group. This is an outer group of fibers which have their anterior termination in the cornea, but which are possibly also related to the circular fibers of the scleral septum. These fibers generally run internal to Schlemm's canal, but occasionally will surround it, so that two or three layers are recognizable on its external surface and six to seven on its internal surface. In most cases these fibers appear to take a crisscross direction. They are usually rather finer in texture than the other fibers of the trabecular meshworks.

2. Fibers of uveal meshwork relating to meridional fibers of the ciliary muscle. This group has one terminal in the most posterior layers of the corneal stroma, frequently extending toward the center of the cornea, well beyond the edge of Descemet's membrane (0.1 mm.). The posterior termination varies. Occasionally all fibers of this group seem to pass into the base of the scleral groove at the tip of the scleral spur, but just as frequently about one-third to one-half of the fibers continues past the tip of the spur to blend with the connective tissue surrounding the meridional fibers of the ciliary muscle. Even in the cases where all fibers seem to enter the tip of the spur, there appears to be some connection, either directly or indirectly by a fibrillary extension, through the spur with the ciliary muscle. There are approximately five layers of fibers in this group. These fibers take the most direct meridional course of any of the trabecular fibers. In meridional sections of the eyes it is common to find one or more fibers shown nearly in its entire length. These fibers are usually somewhat thicker than the corneoscleral ones, and their collagen and Descemet's layer are somewhat more distinctly seen.

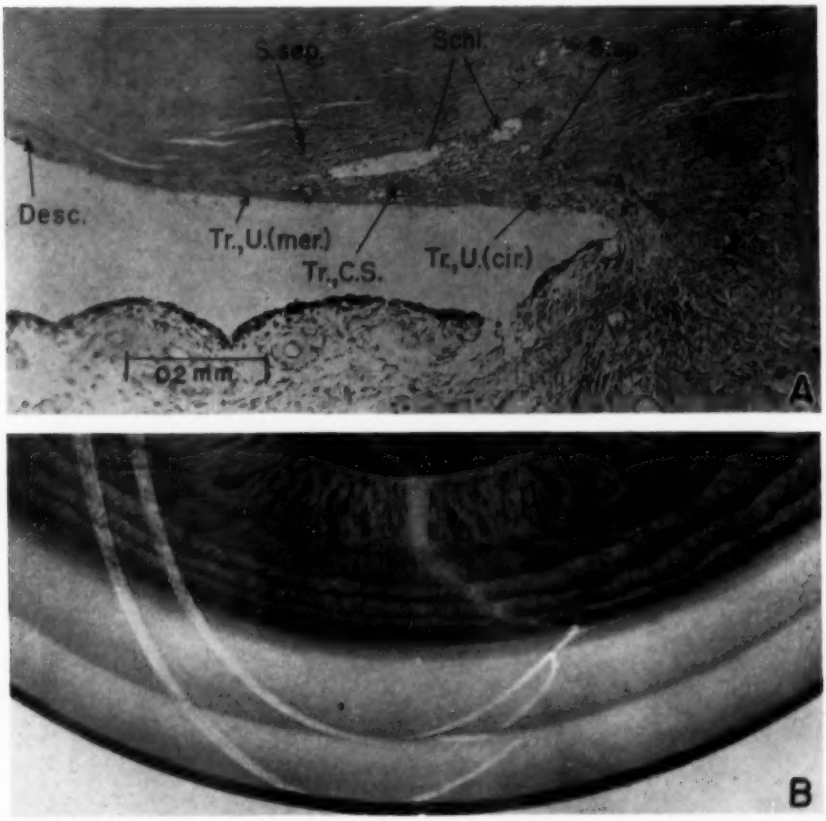
3. Fibers of the uveal meshwork relating to the radial and circular fibers of the ciliary muscle. This next, more internal, group has its anterior termination just behind the edge of Descemet's membrane, at which point the fibers generally take a circular course. The posterior termination of these fibers is typically in the trabeculoconnective tissue sur-

† A more detailed description of these and some other patients is given elsewhere.[9]

rounding the radial and circular ciliary muscle fibers. There are usually three to six layers of fibers in this group.

4. Fibers of the uveal meshwork relating to the root of the iris. This last group of fibers may or may not be present, and in our experience it is entirely absent in many eyes. When present it usually does not form a constant layer, but is represented by isolated fibers or groups of fibers scattered sparsely over the inner surface of Group 3. The position of its anterior termination varies; it may be on the endothelial surface of the edge of Descemet's membrane, on the anterior border-ring of Schwalbe, if one is present, or on the internal surface of the fibers of Group 3, anywhere from the edge of Descemet's membrane to the apex of the angle. Its posterior termination is in the root of the iris, and most frequently it appears continuous with the most superficial layers of the iris stroma. This group usually is one fiber in thickness, and in the eyes in our pathologic collection is never more than two or three fibers thick at the level of the scleral spur. This group may lie in its entire course against the fibers of Group 3, or it may be separated from Group 3 in part of its course, forming a bridge. In the former case it has an endothelial, and sometimes a pig-

Fig. 8.—*A*, histologic section of chamber angle of normal O. D. of Patient F. M.: *Desc.*, Descemet's membrane; *S. sep.*, scleral septum; *Schl.*, canal of Schlemm; *Tr., C. S.*, corneoscleral trabecular fibers; *Tr., U. (mer.)*, uveal trabecular fibers related to the meridional ciliary muscle fibers; *Tr., U. (cir.)*, uveal trabecular fibers related to the radial and circular ciliary muscle fibers; *S. sp.*, scleral spur. *B*, gonioscopic drawing of the same region previous to enucleation.



ment, covering on its internal surface; if it bridges, there is an endothelial covering with associated pigment on its entire surface.

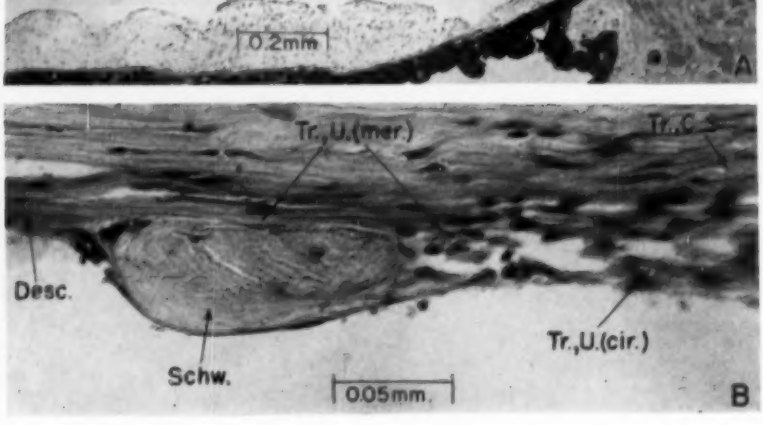
Figure 8*A* shows the histologic section of the chamber angle of a normal right eye, removed as part of an exenteration of the orbit because of carcinoma of the maxillary sinus extending into the orbit. The features of the chamber angle are clearly visible and are indicated in the illustration. The specimen is of particular interest because we had seen the eye gonioscopically prior to its removal (Fig. 8*B*).

B. Anterior Border-Ring of Schwalbe

The anterior border-ring of the trabecular area is commonly described (Schwalbe,[11] Salzmann,[12] and others) as a bundle of fibrillated connective fibers supported by elastic tissue, lying just beyond the edge of Descemet's membrane. This ring is clearly seen in preparations of the chamber angle of many animals (Fritz [13]), but in man it is as a rule not a densely packed group of fibers, but, rather, consists of fibers lying in fairly close proximity. In sagittal sections the fibers are cut across, so that they are viewed end on, and are seen to be separated by slight spaces. There is much variation in this group of fibers. One extreme, not further considered here, is represented by cases in which circular fibers comparable to those in the border-ring are found distributed over the inner surface of the trabecular region, so that they do not constitute a ring. At the other extreme are the cases in which all the circular fibers are gathered into a ring, which may be of such size that it forms an actual prominence extending into the anterior chamber.

When there is a prominent ring, it is made up of closely packed collagen fibers and may contain a greater or smaller amount of elastic

Fig. 9.—*A*, histologic section of chamber angle of normal O. S. of Patient C. B.: *Desc.*, Descemet's membrane; *Schw.*, ring of Schwalbe; *S. sep.*, scleral septum; *Schl.*, Schlemm's canal; *Tr., C. S.*, corneoscleral trabeculum; *Tr., U. (mer.)*, uveal trabecular fibers related to the meridional ciliary muscle fibers; *Tr., U. (cir.)*, uveal trabecular fibers related to the radial and circular ciliary muscle fibers; *S. sp.*, scleral spur. *B*, photograph by oil immersion of another section of the same ring of Schwalbe as that in *A*.



tissue, indicated by small black-staining flecks when the Held-Van Gieson stain is used. Occasionally we have noted a cell nucleus in the bundle, and more rarely still the inclusion of material appearing to stem from Descemet's membrane. With all stains the bundle takes the same deep stain as the scleral spur and the scleral septum, in contrast to the corneal tissue. The ring may appear as a single bundle, or it may be divided into two distinctly recognizable portions. One of the two portions lies usually more internal than the other, and the internal portion tends to surround the other portion at its extremity directed toward the center of the cornea. Ordinarily Descemet's membrane splits just as it reaches the ring, one portion going over the chamber side of the ring, but thinning so as to be soon lost to view; the other portion follows a similar course on the corneal side of the ring. In some cases Descemet's membrane appears only on one side of the ring, either the internal or the external side. From the point where Descemet's membrane stops, the ring is covered only by a single layer of endothelium, identified by the isolated, thin, flat cell nuclei.

Such a prominent ring and its associated chamber angle are reproduced in Figure 9*A*; Figure 9*B* shows a more magnified view of the ring. The two portions of the ring which were mentioned before show up well; however, it contains only a small amount of elastic tissue.

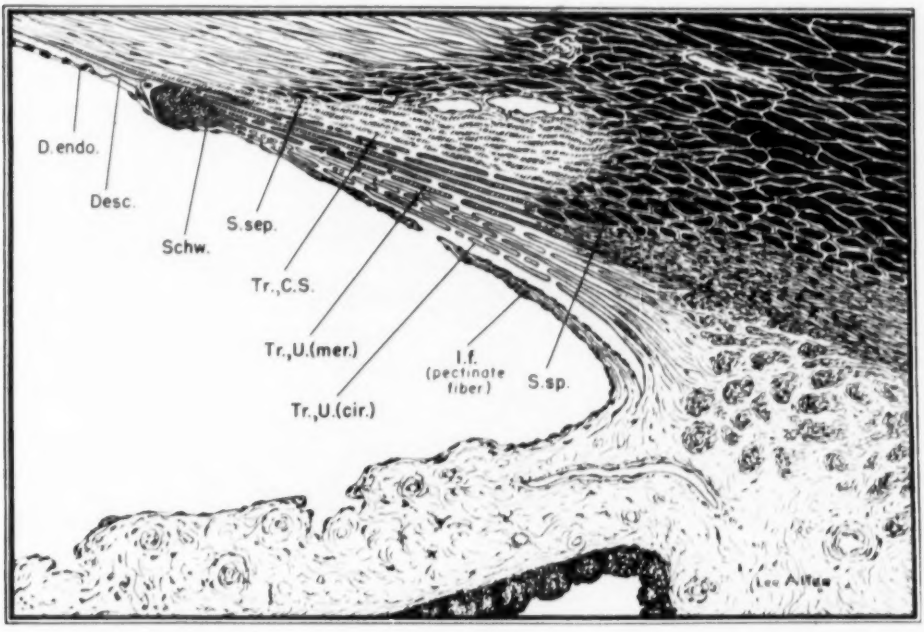


Fig. 10.—Semischematic representation of the chamber angle structures as they frequently relate in the presence of a prominent ring of Schwalbe: *D. endo.*, Descemet's endothelium; *Desc.*, Descemet's membrane; *Schw.*, ring of Schwalbe bulging into anterior chamber; *S. sep.*, scleral septum; *Tr., C. S.*, corneoscleral trabeculum, relating to the spur and septum with a few fibers at corneal end wedging between septum and uveal trabeculum; *Tr., U. (mer.)*, uveal trabecular fibers, one end relating to the meridional ciliary muscle and tip of spur and other wedging between corneoscleral trabecular fibers and Descemet's membrane, some fibers also entering ring of Schwalbe; *Tr., U. (cir.)*, uveal trabecular fibers, one end relating to radial and circular ciliary muscle and other entering ring of Schwalbe; *I. f.*, iris fiber (iris process), inconstant in presence, thickness, and extent upon trabeculum, sometimes bridging as atavistic pectinate fibers; *S. sp.*, scleral spur.

Figure 9 (*A* and *B*) (normal left eye of a 46-year-old man, enucleated because of

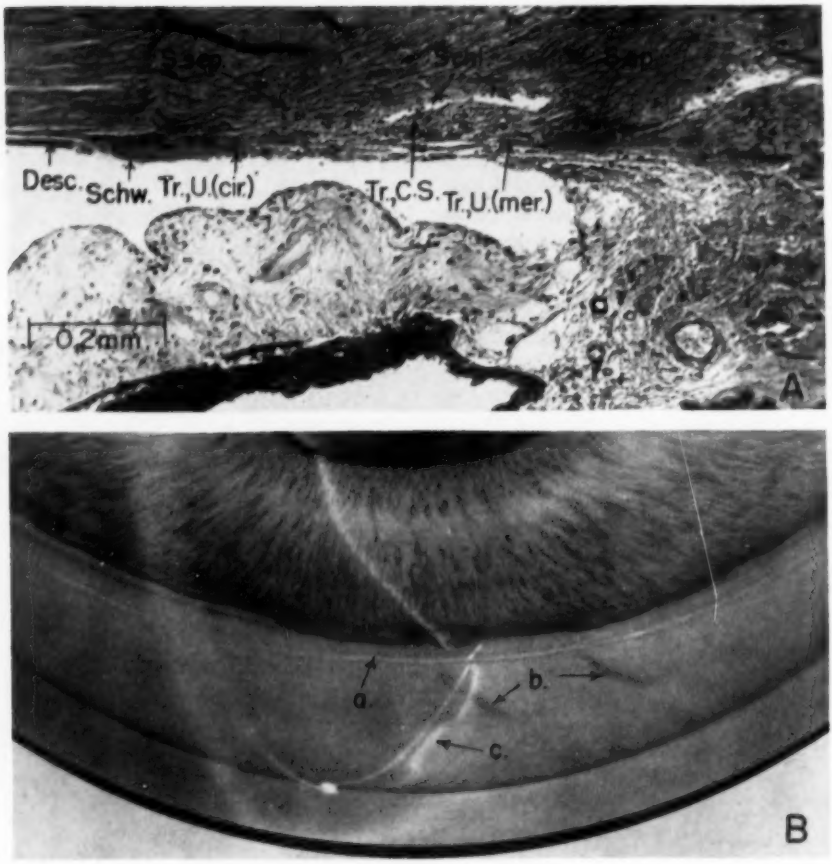


Fig. 11.—*A*, histologic section of upper chamber angle, right eye of a 79-year-old man: *Desc.*, Descemet's membrane; *Schw.*, ring of Schwalbe; *S. sep.*, scleral septum; *Schl.*, canal of Schlemm; *Tr., C. S.*, corneoscleral meshwork;; *Tr., U. (mer.)*, uveal trabecular fibers related to the meridional ciliary muscle fibers; *Tr., U. (cir.)*, uveal trabecular fibers related to the radial and circular ciliary muscle fibers; *S. sp.*, scleral spur. *B*, gonioscopic view of same region previous to enucleation, showing narrowing of the angle due to ciliary body tumor: (*a*) moderately prominent ring of Schwalbe; (*b*) engorgement of two limbal vessels; (*c*) atypical gerontoxon.

epithelioma of the lids and orbit) also shows quite well the connections between the bundle of circular fibers and the trabecular fibers. These connections vary in different cases, but we were able to establish a certain pattern. On the corneal side, fibers belonging to the corneoscleral part of the trabecular fibers (Group 1) are seen, which in our specimens were never incorporated into the ring. Internal to Group 1, one or more fibers of Group 2 may extend about 0.05 to 0.1 mm. beyond the ring, between the corneal stroma and Descemet's membrane. The remaining layers are incorporated into the ring. On the chamber side of the ring, fibers are seen which have their origin toward the inner, or anterior-chamber, aspect of the ring. They lie against its mass and are continuous with the trabecular fibers of Group 3. Trabecular fibers appearing to arise from the posterior part of the circumference of the ring may come to lie either in Group 2 or in Group 3. Also associated in some manner with the ring are the less regular trabecular

fibers of Group 4. These fibers may have their origin on the inner surface of the ring or from its mass. When only one portion of the ring contains elastic fibers, it is always the part from which the fibers of Group 2 emerge. At times the entire ring contains elastic elements, but in our sections these never extended into the inner uveal layers of the trabecular meshwork (Groups 3 and 4).

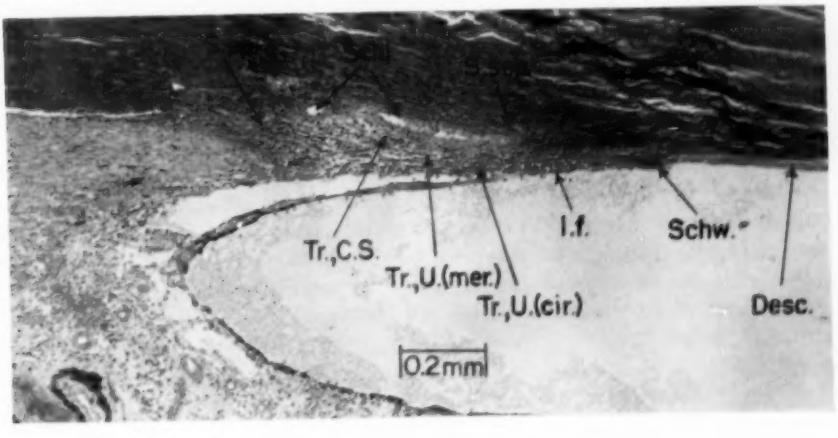
Our findings with regard to the various portions of the trabecular region when a prominent ring is seen in the section are summarized in a schematic drawing of the chamber angle (Fig. 10).

Among the 600 eyes which we examined, we found 72, or 15%, which showed a prominent ring. This is in surprisingly close agreement with our clinical slit-lamp observations. We divided the 72 cases into four groups (1+ to 4+) according to the degree of prominence and noted a moderately prominent ring 11 times, a prominent ring 21 times, a very prominent ring 28 times, and an extremely prominent ring 12 times.

We rated the prominence of the ring in the eye shown in Figure 11*A* as 2+. This is the right eye of a 79-year-old man which was enucleated because of a tumor of the choroid. Prior to enucleation we saw a prominent ring at the periphery of the cornea, in external slit-lamp examination, and obtained a gonioscopic view. In the view of the upper angle, reproduced in Figure 11*B*, the pearly-white line is well visualized. At 12 o'clock there is an anomalous vessel at the root of the iris, and there are two wide, abnormal, obliquely coursing vessels at the external corneoscleral limbus. The unsharp, bulging displacement of the anterior line of the corneal wedge is due to an atypical gerontoxon. The angle shows some narrowing, especially between 11 and 2 o'clock, where the iris root is pushed forward at a point corresponding to the site of the tumor. The histologic section (Fig. 11*A*) goes through this narrowed area of the angle; the forward displacement of ciliary body and iris root is clearly seen.

Another example of a prominent ring, but of one of less pronounced degree (1+), is given in Figure 12. This section (left eye of a 74-year-old man, enucleated because of chronic iridocyclitis with secondary glaucoma following cataract extraction) shows an unusually long trabecular band and also demonstrates the bridging of the trabecular fiber of Group 4, which has been discussed before. The fiber does not lie against the fibers of Groups 3 and 4 at the chamber angle, but

Fig. 12.—Histologic section of chamber angle of left eye of a 74-year-old man with bridging iris process: *Desc.*, Descemet's membrane; *Schw.*, ring of Schwalbe; *S. sep.*, scleral septum; *Schl.*, canal of Schlemm; *Tr., C. S.*, corneoscleral meshwork; *Tr., U. (mer.)*, uveal trabecular fibers related to the meridional ciliary muscle fibers; *Tr., U. (cir.)*, uveal trabecular fibers related to the radial and circular ciliary muscle fibers; *S. sp.*, scleral spur.



rests against a loose meshwork, with resulting open spaces, giving to the angle an appearance comparable to that seen in the eyes of some animals. As will be pointed out elsewhere, we consider this to be an atavistic formation. This bridging fiber, which reaches the fibers of Group 3 midway up the trabecular area, undoubtedly corresponds to the bridging iris processes which we observed gonioscopically.

COMMENT

The accessibility of the trabecular region to external view depends on two or more factors: on the development of the sclera at the limbus, and on how far forward the trabecular region extends, and possibly on additional factors, so far undetermined, regarding a dysgenesis which might limit the extent of the posterior corneal stroma and Descemet's membrane. The relationship between cornea and sclera at the limbus is usually described as being such that the cornea fits like a watchglass into a groove formed by the sclera, in such fashion that the anterior edge of the scleral bevel extends farther forward than the posterior edge, thus effectively blocking the structures of the chamber angle to external view. This description undoubtedly fits the situation in many instances. We have seen, however, that it is not universally applicable. It is not rare to encounter, especially on the nasal and temporal sides, a narrow crescentic extension of the corneal tissue, so that in these areas the outer scleral edge does not reach as far forward as in other parts of the limbus. This was noted and described by Kraupa,[14] who stated that he found it in many eyes and added that in this region the deep layers of the cornea were not clear, but that a striated grayish tissue extended into them. Kayser[15] also reported a case of posterior embryotoxon in which the cornea extended farther peripherally than is normally seen, so that he could survey the chamber angle freely. He gave no description of how it appeared, but Ascher[16] has reported a case of partial coloboma of the scleral limbus which permitted him to visualize Schlemm's canal by external examination. Thus far we have not been favored with finding such an extreme case, and Ascher's case remains unique.

The literature on the "posterior embryotoxon of the cornea" and related structures has been discussed more thoroughly elsewhere.[9] Here we wish only to make a few points.

The "embryotoxon" is more frequent than it would appear from the literature, and the trabecular zone can be observed in every patient by external slit-lamp examination unless there are pathologic changes in the limbal areas.

The strands of iris tissue often seen to insert into the ring of Schwalbe were described by Axenfeld,[1] and he correctly suggested that the whole formation might possibly be termed a rudimentary pectinate ligament in man. These strands are not goniosynechias, as they are called by some authors (Remky,[17] Paufique and others,[18] and Šakić[19]). Neither should the formation be designated as hyaline membrane (Mann[3]), although it does indeed frequently impress one as such on external slit-lamp examination.

The fact that there are no hyaline membranes, but that the formation represents the trabecular zone and a prominent ring of Schwalbe has been established by slit-lamp gonioscopy. Busacca and Pinticart[20] had already observed such a case with the gonioscope and concluded that there was a prominence in the area of the anterior white limiting membrane, in the terminology of these authors. They believed, however, that the prominent line is made up of a thickening of the scleral septum. We think it doubtful that the ring is scleral in origin. While the circular fibers take the same stain as the scleral septum and the scleral spur, and even stain a little more darkly, the anterior layers of the trabecular fibers of Group 2 insert between the scleral septum and the ring in so positive a fashion that there can be no actual connection between the two. Also, the trabecular fibers of Group 3 are continuous

with the fibers within the ring, suggesting that its fibers belong to the trabecular system.

Because of the frequent association of a prominent anterior border-ring of Schwalbe with other congenital anomalies, such as hypoplasia of the iris stroma, corectopia, polycoria, and extensive remnants of the pupillary membrane, the question of its genesis is of some importance. Previous writers have attributed it to an abnormally prolonged contact of the corneal periphery with mesodermal tissue, or to an inadequate absorption of mesodermal tissue during development of the angle of the anterior chamber. Both thoughts are closely connected and derive from the attempt to explain hyaline membranes, hyaline rings, or corneal opacities assumed to exist because of the clinical appearance of the limbus in these cases. Since there actually are no such membranes or opacities present, the theories thus far expressed miss the mark. We have evolved a new concept of the development of the angle of the anterior chamber, according to which this angle opens by cleavage, rather than atrophy, and we consider the prominent ring to be due to an excessive laying down of cells. These views are elaborated and documented in the succeeding paper.[5]

It is difficult to evaluate the connection between the prominent ring and glaucoma. We consider it doubtful that the ring as such is responsible for the glaucoma and think, rather, that where a glaucoma exists it is due to associated anomalies of the iris and chamber angle.

As to terminology, we have already stated that the term "congenital hyaline membranes on the posterior corneal surface" is inadequate and should be discarded. The term "embryotoxon" is meaningless once the nature of the condition is understood and is ambiguous insofar as it has been used by some authors interchangeably for the prominent ring and the faint ring-like opacity formed by the trabecular region. The expression "posterior marginal dysplasia of the cornea," suggested by Streiff,[2] is not quite correct, since one is not actually dealing with a corneal dysplasia, and is even less correct when it is used to include the severer mesodermal abnormalities of iris and chamber angle. In this respect the designation "dysgenesis mesodermalis corneae et iridis" of Rieger[21] or, if one accepts Hagedoorn's criticism,[22] "dysgenesis mesostromalis corneae et iridis," would seem to be preferable, although they might seem somewhat formidable for the frequently seen simple form of a prominent ring. We are aware that severe abnormalities may be associated with it and that the trabecular zone may be abnormally wide and the ring very prominent, but we have no single term to offer to cover all abnormalities. We suggest that these structures be called what they actually are: the trabecular zone and a prominent line of Schwalbe.

SUMMARY

The trabecular region is visible at the periphery of the posterior corneal surface in almost every person in external slit-lamp examination, appearing in broad direct focal illumination as a band of grayish, translucent tissue showing fine crisscross or regular striations. This band considerably differs in width in different persons and in different regions of the limbus. This same tissue appears to have a "golden glow" when studied with a very narrow slit-lamp beam.

In about 15% of all patients examined, the trabecular region ended corneaward in a more or less prominent whitish line, often bearing clumps of pigment.

This line was identified by gonioscopy as a prominent anterior border-ring of Schwalbe.

A prominent anterior border-ring is often associated with mild to severe hypoplasia of the anterior stromal layer of the iris and other congenital anomalies, such as corectopia, polycoria, etc. It is congenital and familial.

A prominent ring may be associated with glaucoma, but it is our impression that the ring itself is not causally related to the glaucoma.

Histologic studies of the trabecular region are presented. Among 600 sectioned eyes a

prominent anterior border-ring was seen 72 times, and its histologic features are reported.

With regard to the genesis of the prominent ring, it is our conclusion from embryologic investigations that it is due to an excessive laying down of cells and excessive relative growth, not to a lack of resorption of mesodermal tissue. Abnormal iris processes bridging from the iris root to the trabecular meshwork and prominent ring are an atavistic formation.

"Congenital hyaline membranes at the posterior corneal surface," as applied to the formations described in this paper, do not exist, and this term should be dropped. The term "embryotoxon" is ambiguous and superfluous; "posterior marginal dysplasia of the cornea" is not an entirely correct designation; "dysgenesis mesodermalis (or mesostromalis) corneae et iridis" is better, but we suggest that the structures under consideration should simply be referred to as "trabecular zone" and "prominent anterior border-ring of Schwalbe."

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A New Concept of the Development of the Anterior Chamber Angle

Its Relationship to Developmental Glaucoma and Other Structural Anomalies

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In a previous paper [1] we reported the identification of the embryotoxon corneae posterius, Axenfeld, or glassy-appearing membranes at the periphery of the posterior corneal surface, as the trabecular meshwork and the anterior border-ring of Schwalbe. In the cases available to us for complete study this identification was done through external and gonioscopic examination and by histologic diagnosis. Further assurance was gained by a careful histologic study of the chamber angle region of 600 eyes in our pathology collection. We also searched for new facts about the structure of the normal chamber angle to serve as basis for the interpretation of abnormal appearance in clinical cases. This led, in turn, to developmental and comparative anatomic investigation,* some of which will be reported here and some in a succeeding publication.[2]

A new concept of the manner in which the chamber angle develops formulated itself as evidence from the separate studies was integrated. It began to appear that the chamber angle does not open by a process of atrophy with resorption, as stated in the accepted theory, but, rather, opens by a simple separation of two dissimilar layers of mesodermal tissue. Formerly we had assumed with others that a measurable quantity of embryonic mesoderm must be eliminated from between the trabeculae and the iris root in order that the chamber may be extended to the scleral spur region. The new theory that the angle opens by a cleavage required that we find the mechanical forces which could accomplish this action. We sought causes in changes in form, volume, and space relationships, and in the dynamics of growth from the fetal eye to the fully developed adult eye. We found that simple geometric relationships and the factors influencing the three-dimensional growth readily explain the pulling open of the chamber angle.

Verification of these new findings cleared the way for a better understanding of conditions in which so-called mesodermal remnants were thought to play an important part. It should clarify the structural situa-

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* All histoanatomic studies were done on tissue sections 10μ in thickness. Sections stained by the Held, Held-Van Gieson, and Mallory methods, besides those stained with hematoxylin and eosin, were available for nearly all eyes. The recognition of cell groups and their differential concentration, character, and boundaries; of size and characteristic appearance of cells within each group, and of cleavage lines and points of insertions between groups is dependent upon the thinness of the sections and upon differential stains. Many of the observations recorded cannot be made in thick sections, especially in fetal eyes, where there is so little space between cell nuclei in the mesodermal tissue that the compactness obliterates delicate, but important, features. Mrs. K. E. Lindorfer made the preparations of routine histopathologic specimens, of consistent excellence.

tions in developmental glaucoma, and it might eventually lead to improved methods of dealing with these entities, either surgically or pharmacologically.

The most important observations and conclusions from our studies may be listed as follows:

1. The chamber angle normally opens by a simple splitting apart of two distinct layers of mesodermal tissue. On the anterior (external) side of the plane of separation, the cells of the trabecular tissue clearly have a differential concentration and architectural arrangement, even before cleavage of the chamber angle begins. As may be judged from the structure of the normal adult trabeculae, these cells are so constituted that they will not adhere along their sides to adjacent cells, but, for structural purposes, adhere at their ends to form long fibers and stellate junctions. In this way, the spaces between the fibers are created and maintained. The innermost layer of cells (those nearest the anterior chamber) follows the same rule, and, therefore, cannot adhere to the iris and ciliary body tissue, against which it had rested during early fetal life. Only abnormally oriented or differentiated cells may adhere to the latter tissues.

Posterior to the plane of separation, the iris and ciliary body cells adhere in a different way to each other, so that they normally form a more homogeneous mass, which can separate as a unit from the adjacent trabecular tissue.

The opening of the angle by cleavage coincides with a period of unequal growth within the region.

2. The number of cells laid down in the embryo and fetus, their position, orientation, differentiation, and growth, largely determine the appearance and functional efficiency of the chamber angle tissues. Some of these factors are easily demonstrated. Other factors which might have to be taken into account, such as improper timing in the development and abnormal environmental conditions, have not had adequate consideration in the present investigation.

3. Extensive unequal growth is a major force responsible for the opening of the chamber angle. The magnitude of increase in volume of the chamber angle tissues is greater than may be generally realized, and during this period marked changes in spatial relationships are achieved.

4. In the normal, fully developed chamber angle borders of cell groups tend to be distinct both gonioscopically and histologically. The fibers in the human trabecular meshwork tend to have constant thickness and clear boundaries, and their reticulation has an almost geometric character.

5. In many congenitally glaucomatous eyes, the boundaries of the cell groups are less distinct, the cells of the trabeculae are less regularly organized into fibers, and the reticular pattern is less geometric and more diffuse and homogeneous in appearance than in normal eyes. This suggests poor differentiation. The inner uveal trabecular fibers are frequently only one-half to two-thirds as long as those in the normal chamber angle, and the base of the trabecular area is somewhat thicker than normal. Schlemm's canal and the surrounding corneoscleral tissues are often extremely abnormal. Schlemm's canal may be absent and the region filled with sclerotic tissue. In most of these eyes the cleavage between the trabecular meshwork and the ciliary and iris bodies has not been completed. Careful study shows no demonstrable total excess of tissue, but, rather, an imperfect organization and distribution.

6. Prominent anterior border-rings of Schwalbe and pectinate ligament features, present in some anomalies, are not the result of incomplete atrophy. They have positive structural characteristics and are usually accompanied by a high degree of organization of the trabecular meshwork and of the cells in Schwalbe's ring. Special groups of cells, strategically located, which create these prominent features can be seen in fetal eyes.

7. Combinations of two foregoing situations (5 and 6) do occur; therefore they should not be thought of as exact opposites.

Each consists of several digressions from what we have considered normal. One must, therefore, expect to find a wide variety of each and varieties in combinations of them.

8. Since no measurable amount of tissue is lost during chamber angle development, and the excessive or abnormal forms seen are not "remnants," such a term as "mesodermal remnants" or "persistent fetal tissue" should be discarded.

ATROPHY THEORY OF DEVELOPMENT OF THE ANTERIOR CHAMBER

The term "atrophy" has been used to describe the process by which the chamber angle extends backward, exposing the trabecular meshwork for efficient drainage of aqueous. It is somewhat difficult to tell the exact meaning attached to the term by the various authors. Since it is nearly always used together with the terms "resorption," "disappearing," "absorbed," etc., we are led to believe that it is used to indicate the loss of some detectable quantity of a tissue which has been laid down in the embryo to serve no known useful purpose, and which must degenerate and be eliminated during fetal life. We have not been able to read into the descriptions of atrophy the mere differentiation and parting of cell layers without some loss of tissue.

Much of the thinking on congenital glaucoma has been based upon the belief that remnants of embryonic mesoderm are sometimes present to fill the filtration angle and thereby block the passage of aqueous out of the anterior chamber into the venous systems.[3] It is not explained why the tissue should be there in the first place, or how the supposed atrophy can come about and progress just to the desired limit in the normal eye.

The "atrophy" and "remnant" theory has been the rationale behind the choice of surgical procedures and the invention of new procedures and techniques in the treatment of congenital glaucoma.[4] There is now an increasing accumulation of evidence and opinion that infantile glaucoma and surgical failures are due to other pathologic, congenital, or developmental anomalies than mesodermal remnants.† Perhaps it will be shown that goniotomy, goniopuncture, etc., succeed for reasons similar, but not identical, to those hitherto considered to be correct.

If one can demonstrate that no mesodermal remnants are involved in congenital glaucoma, and if the literature can be relieved of the misleading concepts of "atrophy" and "remnants," new efforts can be made toward finding the causes of developmental glaucoma.

From Seefelder's time onward,[5] all investigators appear to have interpreted an area of loose, mesh-like mesoderm, lying between the developing trabeculae and apparently the iris and ciliary body, as a tissue undergoing atrophy. Anderson,[3] with whom we agree on much of the morphology of the trabecular tissue and interpretations of comparative anatomy findings, still subscribes to the theory of "atrophy" and "remnants" in his monograph on congenital glaucoma. As late as 1947, Ida Mann accepted this theory in describing the development of the angle. In her chapter in Troncoso's book on gonioscopy,[6] Mann states:

> Probably from a practical point of view the most important phase of development of this region is the process of atrophy of the mesoderm at the iris root. The amount to which this takes place in different individuals determines very largely both the gonioscopic appearance and the functional efficiency of this important region.

No doubt hereditary factors are most fundamental in determining the number, position, orientation, and differentiation of the cells in this region, and thus in determining the development and final form of the area, subject only to abnormal conditions in the embryonic and fetal environment. Evidence of heredity is difficult to establish in normal angles, but is found in deviations from the normal, as in angles containing prominent rings of Schwalbe and rudimentary pectinate ligaments.

† Shaffer, R. N.; Maumenee, A. E., and Scheie, H.: Personal communication to the authors.

HISTOLOGIC EVIDENCE OF CLEAVAGE

Our collection of fifth- and sixth-month fetal eyes contains many sections where the appearance in the plane of the developing chamber angle is that of a loose meshwork. The tissue looks exactly like that in published photographs, purporting to show the "resorption" type of atrophic process.

After finding a number of fetal chamber angles which, at similar stages of development, contained none of this "loose meshwork," we felt the need for a new interpretation of this appearance. After further study, we believe that the loose meshwork represents tissue under tension in a situation where a few cells on one side of the cleft adhere abnormally to the opposite tissue. We believe that in many cases this is due to abnormal cell orientation and slow or faulty differentiation, but that in most of the nearly normal eyes these adhesions are tenuous, and fail under further increase in tension. However, adhesions may persist throughout life in scattered areas of some eyes, and over the entire circumference of the chamber angle in a few.

If an adhesion is strong, but only two or three cells are coupled together, it will bridge the angle until tension breaks the strand after the cells have exhausted their ability to lay down collagen. Where the break is near the trabeculae, not much, if

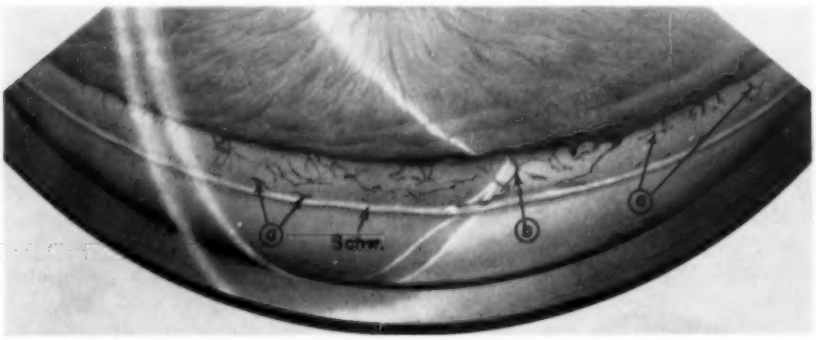
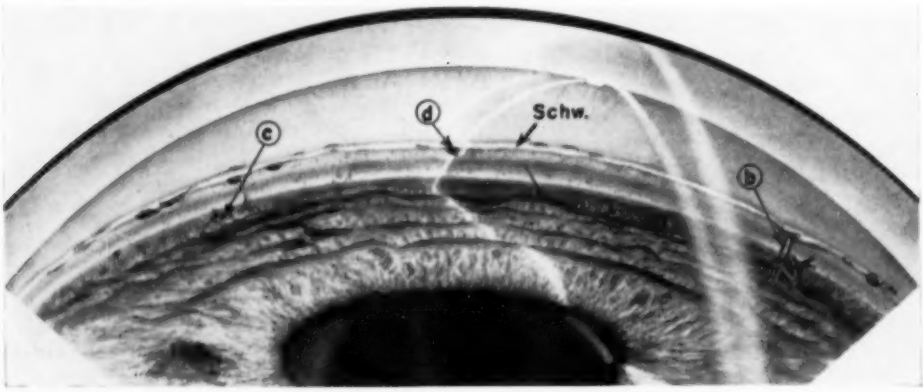


Fig. 1.—Upper quadrant of chamber angle O. D. of Patient R. L., a man, aged *36*. *Schw.*, prominent anterior border-ring of Schwalbe; *a*, pigmented fibers detached from the iris and adherent to the trabecular band; *b*, one of several pectinate ligament strands bridging the angle.

Fig. 2.—Lower quadrant of chamber angle O. S. of Patient M. B., a man, aged *29*. *Schw.*, prominent anterior border-ring of Schwalbe; *b*, pectinate ligament strands; *c*, iris processes; *d*, one of several clumps of heavily pigmented iris tissue separated from the iris during cleavage and adherent to the ring of Schwalbe.



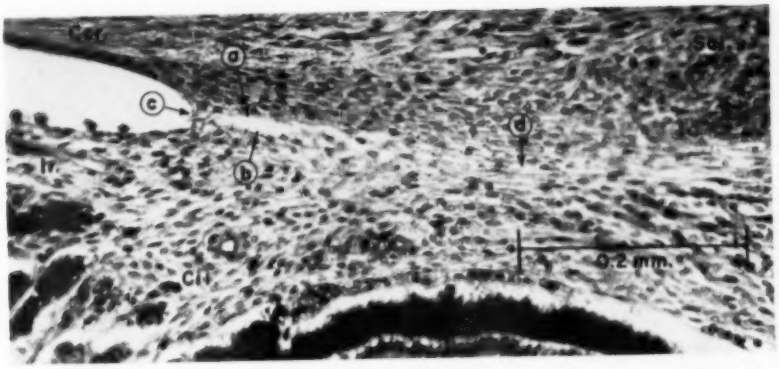


Fig. 3.—Section (Mallory stain) through the chamber angle of a 220 mm. fetus. Cleavage is occurring first between the inner surface (*a*) of the uveal trabecular tissue and the anterior surface (*b*) of the iris-ciliary body mass. The apex of the fetal angle (*c*) will separate next. The cleft will later extend at least as far as *d*, a few cell layers internal to and slightly beyond the scleral spur. *Ir.*, iris; *Cil.*, ciliary body; *Cor.*, cornea; *Scl.*, sclera.

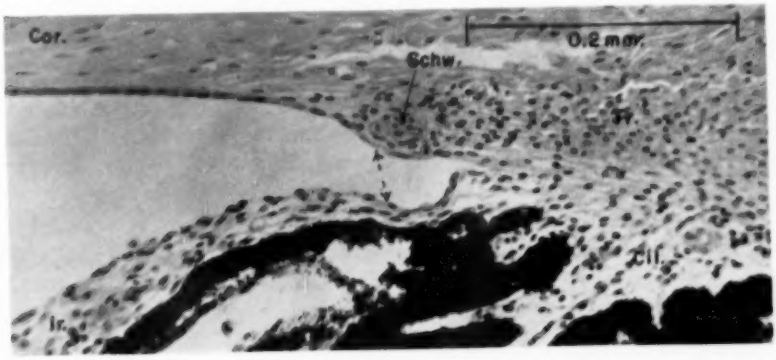
any, evidence will be seen in the full-grown eye. Where the break is near the iris or ciliary body, thread-like fibers, sometimes pigmented, may be seen gonioscopically on the adult trabecular zone, as demonstrated at *a* in Figure 1.

Rather often cells are so coupled in these bridging threads that they will not break. In these cases pectinate fibers (Troncosco[6]), or iris trabeculae, or iris processes (Busacca[7]), can be seen in the adult angle, as shown at *b* in Figures 1 and 2. If these threads adhere along their length to the corneoscleral side of the angle, and do not extend far beyond the ciliary body band, as at *c* in Figure 2, they constitute what Busacca[7] calls the basal band of the iris processes. Troncosco[6] and others think of all these iris threads as being related to the animal pectinate ligament, an opinion in which we concur, as we shall discuss later.

In rare cases, thick clumps of iris tissue, such as shown at *d* in Figure 2, are torn away from the iris and left upon the trabeculae and anterior border-ring of Schwalbe.

In some sections from fifth- and sixth-month fetal eyes there is no "loose mesodermal meshwork." The tissue on each side

Fig. 4.—Section (hematoxylin-eosin stain) through the chamber angle of a 160 mm. fetus (from the collection of Shaffer and Maumenee, reproduced by permission). Cleavage has progressed in a particularly clean-cut manner beyond the prominent ring of Schwalbe (*Schw.*). Note the sharply outlined imprint of the ring (*Schw.*) upon the root of the iris; *Tr.*, developing trabecular fibers; *Cil.*, ciliary body; *Ir.*, iris; *Cor.*, cornea.



of the potential chamber angle is compact, and the various layers can be studied. No sign of disintegration is apparent in these tissues. Only a clean, simple splitting is taking place along a line between the trabecular zone and the ciliary body.

A question as to whether the separation might, in accordance with the cleavage theory, continue far enough to separate the ciliary body from its insertion into the scleral spur is not valid. The plane of separation passes several cell layers internal to the scleral spur, thus avoiding its possible disinsertion.

PATTERN OF CLEAVAGE

The opening of the angle by cleavage follows the rather typical pattern indicated on Figure 3. Where no excessive cells are present to form prominent rings of Schwalbe or pectinate strands, the schism occurs exactly along the inner layers of the uveal trabeculae (Fig. 3, *a*). The first separation is generally seen over the massive portion of the fetal ciliary body (Fig. 3, *b*), which is developing in a different direction and at a different rate from the trabeculae. In many sections of fetal eyes there is adhesion to one or more cells exactly at the apex of the fetal chamber angle (Fig. 3, *c*). This may be due partly to the presence of factors tending slightly toward the formation of a pectinate ligament, since this is the position in which it is found in other mammals. These adherent cells eventually separate, allowing the cleft to unite with the fetal anterior chamber. At this time the peripheral one-half to one-third of the trabecular meshwork is still in contact with the ciliary tissue. The cleft then extends gradually beyond the scleral spur, in the region marked *d* in Figure 3. As will be pointed out later, the failure of this latter part of the cleavage to take place may account for some of the features present in cases of congenital glaucoma.

The cleavage does not always begin over the region of the ciliary body. An extremely neat and clear-cut cleavage was present in a 160 mm. fetus from the collection of Shaffer and Maumenee (Fig. 4), reproduced by permission. Here the cleavage is beginning at the apex of the fetal angle, the approximate original location of which is indicated by the dotted line; a well-formed ring of Schwalbe containing many nuclei has separated from the root of the iris, where it has left a deep imprint; the line of future cleavage can readily be discerned.

CELL LAYER COUNT AS EVIDENCE AGAINST THE THEORY OF ATROPHY

The number of cell layers between the rudimentary Schlemm's canal and the pigment epithelium of the ciliary body in the fetal eye was compared with that in the same structures in the adult. Figure 5*A* is a section from a 120 mm. fetus just prior to the opening of the chamber angle. The potential cleft is seen as a nearly straight clear line passing from *a*, at the apex of the fetal angle, through *c* to the potential apex of the adult angle, at *b*. There are approximately 12 layers of cells between the rudimentary Schlemm's canal, at *d*, and the cleft *c*. There are approximately 15 layers between *c* and the ciliary epithelium, at *e*.

In the adult angle of Patient E. E., pictured in Figure 5*B*, the ciliary body is in a somewhat forward position, so that the formerly adjacent mesodermal tissues are not too far separated for easy identification. Cell layers comparable to those in the fetus can be counted with assurance. There are 15 layers between Schlemm's canal, at *d'*, and the inner surface of the trabecular tissue, at *c'*. There are approximately 25 layers between *c'*, on the root of the iris, and *e'*, at the ciliary epithelial surface.

Similar comparisons were made between fetal and normal adult eyes in which more care was needed in identifying the iridociliary tissue originally adjacent to the trabeculae. The averages indicate that there is an increase of several cell layers, instead of any decrease.

Thin iris roots seen in some adults have no fewer cell layers than can be found in the comparable region of many fetal eyes. It can be assumed that in such adult eyes the

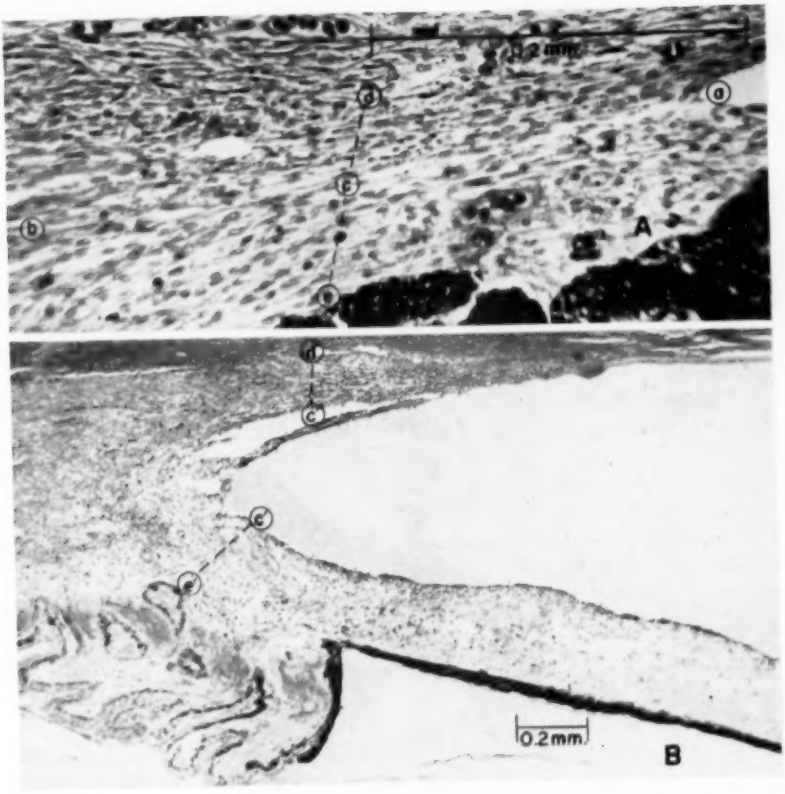


Fig. 5.—*A*, section (Held stain) through the chamber angle of a 120 mm. fetus (*a*) apex of the fetal angle; (*b*) predicted apex of the adult angle. Cleavage can be predicted along a visible line from *a* through *e* to *b*. Approximately 12 layers of trabecular fibers can be counted between the potential cleft, at *e*, and Schlemm's canal, at *d*. Approximately 15 layers of cells can be counted in the iris-ciliary body tissue between *e* and *e*.

B, section (Held stain) through the chamber angle O. D. of Patient E. E., a man, aged 74. Approximately 15 layers of trabecular fibers can be counted between *c'*, at the inner surface of the meshwork, and *d'*, at Schlemm's canal. Approximately 25 layers of cells can be counted between, *c'*, at the anterior surface of the iris-ciliary body tissue, and *e'*, next to the ciliary processes in an area comparable to that between *e* and *e* in *A*.

iris root was thin to start with and that little thickening took place in that region during the period of proliferation in other parts of the uvea.

VOLUME AND FORM CHANGES AT THE CHAMBER ANGLE DURING GROWTH

The growth processes in the region of the chamber angle include differentiation, proliferation, cell growth, and cavitation, with its attendant fluid filling. The special requirements placed upon each of these processes, and variations in the amount of each in different regions, contribute to change of form during growth.

The amount of volume increase still to be achieved in the entire eye, and therefore in the chamber angle region, after the fourth fetal month may not be universally recognized. A full understanding may help toward appreciating the extent of the proliferation, cell growth, and cavitation required to bring the structures to their final form and efficiency, and will easily show that atrophy is

not a requirement for the opening of the chamber angle. We shall, then, consider first the size increases as a suitable background against which to judge the specific growth processes.

MAGNITUDE OF GROWTH OF CROSS-SECTIONAL AREAS OF THE ANGLE

In histologic cross sections, which are essentially two-dimensional, the magnitude of size change during development is only partly evident. However, some conception can be derived from comparing the fetal and the normal adult structures, as done in Figure *6A* and *B*. The rudimentary scleral spur can be seen in the 120 mm. fetal eye as a condensation of tissue at *S. sp.* (Fig. *6A*), although scattered deep-staining circular fibers are present even much farther forward. The potential anterior border-ring of Schwalbe can be predicted at *Schw.*, although the most external uveal trabecular fibers extend farther forward, as indicated on the Figure. The distance between the anterior border-ring and the scleral spur in the adult (Fig. *6B*) is two and one-half times as great as in the fetal eye (Fig. *6A*). The increase in area equals approximately the square of this distance. In other words, the cross-sectional area had increased 6.25 times.

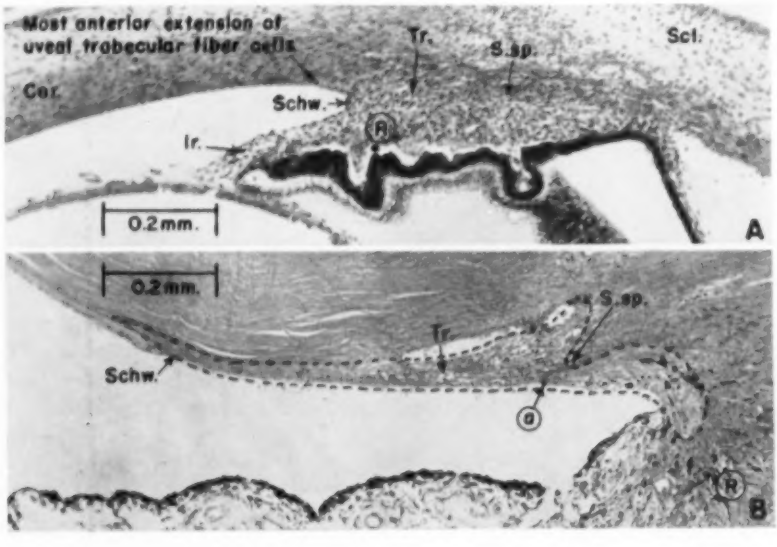
This increase in cross-sectional area is well appreciated upon noting that almost the entire cross section of the trabecular meshwork, ciliary body, and iris of the eye of the 120 mm. fetus shown in Figure *6A* could be enclosed within the dotted outline of the adult trabecular region alone, shown in Figure *6B*.

INCREASE IN TOTAL VOLUME

Some concept of the volume increase may be gained from Figure 7, in which the sizes of the combined ciliary body and iris of a fifth-month fetal eye are compared with the

Fig. 6.—*A*, section (Held-Van Gieson stain) through the ciliary body of a 120 mm. fetus: *Schw.*, rudimentary anterior border-ring of Schwalbe; *Tr.*, trabecular cells; *S. sp.*, rudimentary scleral spur; *R*, region to be occupied later by the radial and circular ciliary muscle tissue; *Ir.*, iris mesoderm; *Cor.*, cornea; *Scl.*, sclera.

B, section (hematoxylin-eosin stain) through the chamber angle of normal right eye of Patient, F. M., a man aged 57. Labels corresponding to those in *A* indicate the vast amount of growth which must occur between these two stages. *R*, fully developed radial and circular ciliary muscle region; *a*, unusual posterior position of the anterior insertion of inner uveal trabecular fibers.



ciliary body only of a normal adult, Patient D. J. The eye has increased about 35 times in volume, and, while it is difficult to estimate the volume of the ciliary body with its irregular ciliary processes, its increase is assumed to be approximately in the same proportion.

During the change from the smaller to the larger volume a marked inequality of growth is evident in the chamber angle region. While this is not emphasized enough by earlier writers, it is hinted at in their statements that the major circle of the iris is anterior to the level of Schlemm's canal early in development, and that normally it arrives at a point behind the scleral spur in the adult (Mann[8]). Unless the mass of the ciliary body were to shrink backward in the eye, such a change could be accounted for only by inequality of growth.

It is misleading to select the scleral spur as a fulcrum or fixed point around which the growth and the changing relationships can be described, as was done by some authors. Figure 8 shows that the geometric center of the eye would be a better arbitrary reference point to which growth changes might be related.

SOME ASPECTS OF INEQUALITY OF GROWTH

Diagrammatic concentric cross sections of the fifth-fetal-month eye and the adult eye are represented in Figure 8*A*. The proportions on which these cross sections are based and the measurements mentioned in the following considerations were obtained from two fifth-month fetal eyes and two normal adult eyes.

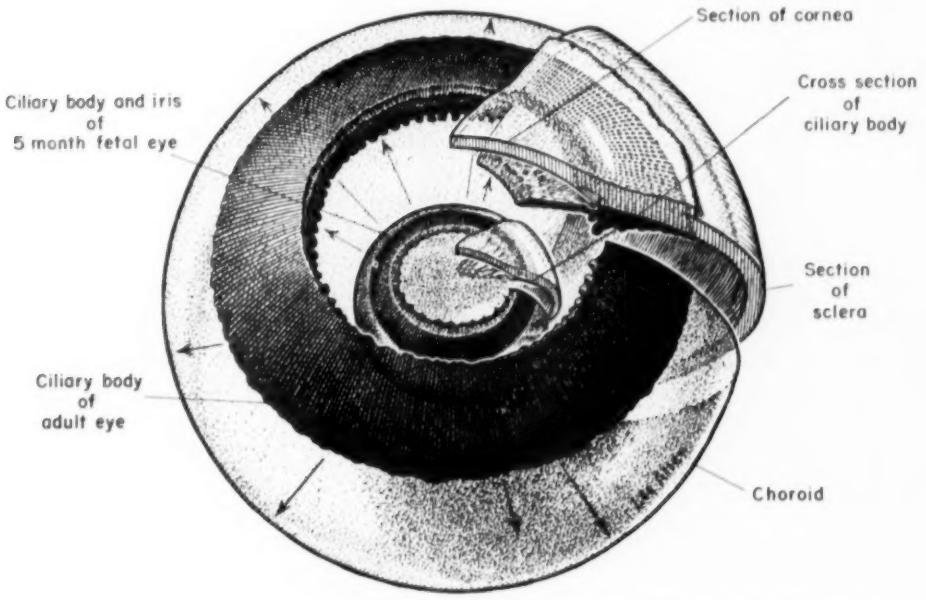

Section of cornea
Cross section of ciliary body
Ciliary body and iris of 5 month fetal eye
Section of sclera
Ciliary body of adult eye
Choroid

Fig. 7.—Schematic drawing showing the fifth-month fetal eye placed concentrically within the normal adult globe, to illustrate the 35-fold increases in total volume between the two stages.

The diagram makes clear the changes which occur in the topography of the iris and ciliary body, and which suggest the forces required to open the chamber angle by cleavage.

The external surface of the ciliary body, indicated between *a* and *b*, grows four times in length, while side *b-c* increases 2.9 times and side *c-a* only two times. Point *c'* would, therefore, appear to have moved backward in the adult eye, and side *a'-c'* to have changed

its position by having rotated almost 80 degrees. A somewhat more accurate description might state that point *a'* has grown outward and forward around point *c'*, but only relatively, since point *c'* has also grown outward, and possibly forward.

The change in the cross section of the ciliary body is shown even better in the purely geometric simplification in Figure 8*B*. Points *a*, *b*, and *c* represent the apices of the triangular cross section of the fetal ciliary body; points *a'*, *b'*, and *c'* are comparable points in the adult eye. The growth ratios used above were also applied in Diagram 8*B*.

The inequality of growth is further diagrammed in Figure 8*A* by the dot-and-dash lines radiating from the center of the two concentrically arranged cross sections. If growth were equal throughout the eye, radial lines passing outward through the ora serata, the scleral spur, and the anterior tip of the ciliary processes of the fetal eye would indicate that corresponding structures in the adult eye should grow in the direction of points *d*, *e*, and *f*, respectively. Instead, these structures have undergone an unequal displacement toward *d'*, *e'*, and *f'* during growth, as indicated by the arrows at *d*, *e*, and *f*.

GROWTH PROCESSES CONTRIBUTING MOST TO UNEQUAL GROWTH AND CLEAVAGE OF THE CHAMBER ANGLE

Proliferation

Proliferation occurs in the ciliary body and iris well beyond birth, but, especially

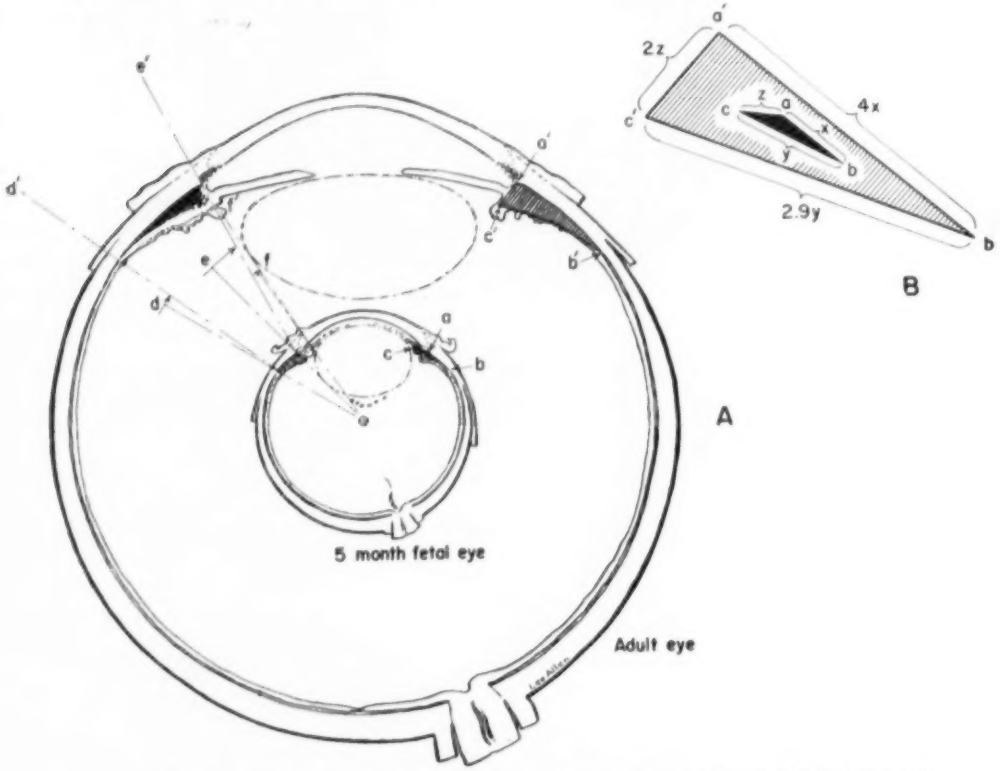


Fig. 8.—*A*, concentrically arranged schematic cross sections of the fifth-month fetal eye and the normal adult eye, showing inequalities of growth of structures, as related to the anatomic centers of the globe.

B, superimposed cross sections (reduced to basic geometric forms) of the ciliary body areas of the fifth-month fetal and normal adult eyes, showing different growth ratios of the major surfaces.

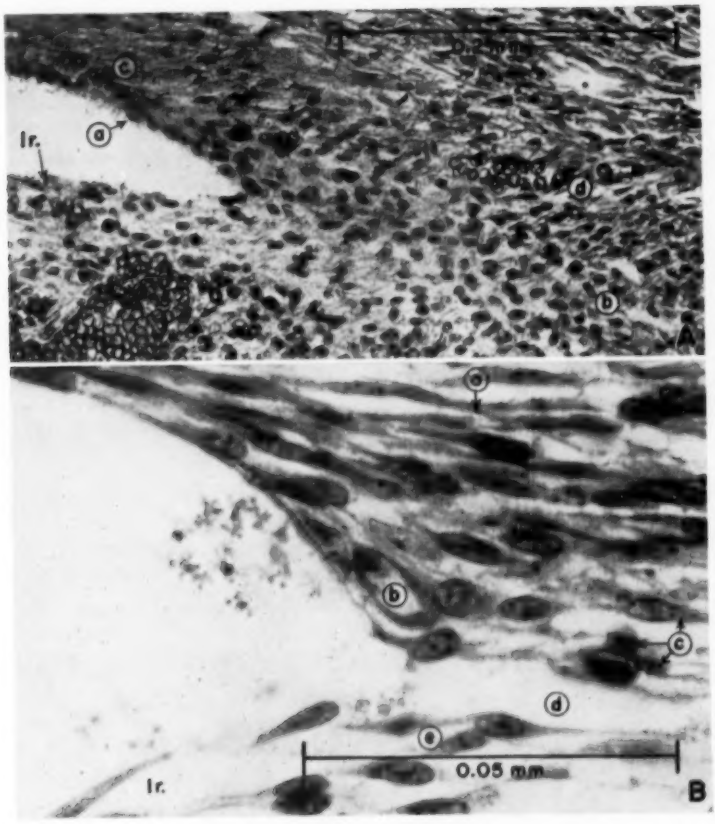


Fig. 9.—*A*, section (hematoxylin-eosin stain) through the chamber angle of a 120 mm. fetus. Large granular nuclei of the uveal trabecular cells are seen in the region between *a* and *b*. The corneoscleral trabecular fibers are developing between *c* and *d*; *Ir.* iris.

B, section (Held stain) through the chamber angle of a fifth-month embryo: (*a*) Region in which moderately developed corneoscleral trabecular fibers are seen; (*b*), deep-amber-staining collagen fibers in a spiral arrangement suggesting a rudimentary, minimal anterior bordering ring of Schwalbe; (*c*), deep-amber-staining collagen of the developing uveal trabeculae; (*d*) cleft beginning between the trabecular tissue and the iris-ciliary tissue, at *e*; *(Ir.)* iris.

from the fourth fetal month onward, it aids in forming the chamber angle by altering the mass relationships within the uvea. Its most noticeable contribution is perhaps related to the development of the radial and circular bundles of the ciliary muscle. These are not present at the fourth-month stage, and their subsequent growth greatly changes the contours of the ciliary body. This is clearly shown in Figure 6. Whereas there are no bundles in the fetal ciliary body, *R*, in Figure 6*A*, they are well developed in area *R* of Figure 6*B*. After the fourth month the increasing mass of these bundles is effective in moving the ciliary processes and the iris root farther and farther from the trabecular region. The result is that the ciliary tissues, formerly resting against the uveal trabeculae, are pulled away, thus helping to extend the chamber outward and backward.

Proliferation is not recognized with certainty in the trabecular region itself after the fifth fetal month, although this might be the manner in which the outer coats of the trabecular fibers are created. The endothelium of Descemet's membrane may need to

proliferate outward and then around the collagen strands of the trabecular fibers. On the other hand, cells similar to those of Descemet's endothelium may be endogenous here. For the rest, the trabecular fibers appear to enlarge their mass by cell growth.

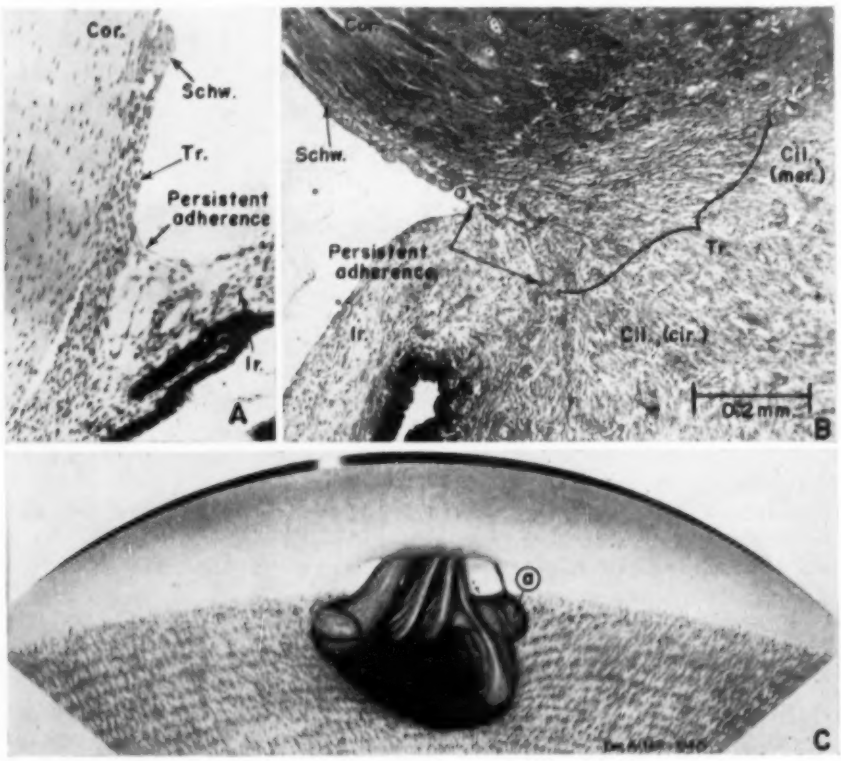
Cell Growth

Figure 9*A* is a high-power view of the histologic section of the chamber angle of a 120 mm. fetus. A concentration of large granular cell nuclei, which are differentiated and are ready to lay down the collagen fibers of the uveal trabeculae, can be recognized between points *a* and *b*. The cells of the corneoscleral portion of the trabecular meshwork appear to have become active in this manner earlier, because the rudimentary meshwork can be seen to exist in the area between *c* and *d*. Here the nuclei are smaller than in the uveal layer, but they are still granular in appearance. It is difficult to tell

Fig. 10.—*A*, section (hematoxylin-eosin stain) through the chamber angle of a fetus of approximately 8½ months (from the collection of Shaffer and Maumenee, reproduced by permission). Persistent adherence of the peripheral iris to the trabecular tissue is indicated. *Schw.*, prominent anterior border-ring of Schwalbe; *Tr.*, trabecular tissue; *Cor.*, cornea; *Ir.*, iris.

B, section (Mallory stain) through the chamber angle O. D. of Patient M. M., a woman aged 18, with congenital glaucoma. The persistent adherence of the iris root to the trabecular tissue is indicated. Poorly differentiated and poorly organized trabecular tissue is seen in the bracketed area (*Tr.*). *Schw.*, anatomic position of the anterior border-ring of Schwalbe; *Cil. (Mer.)*, meridional ciliary muscle fibers; *Cil. (Cir.)*, region of the circular ciliary muscle bundles; *Cor.*, cornea; *Ir.*, iris.

C, upper quadrant of chamber angle O. S. of Patient M. M., for comparison with the histologic section of O. D. Drawing inverted from actual view in gonioprism mirror. The trabecular band is largely hidden by persistent adherence of the iris root. Ciliary processes at *a* appear to be abnormally far forward. Other ciliary processes are incarcerated in a trephine opening.



without a comprehensive study whether there are in a given specimen just enough, too many, or too few cells present in the whole trabecular region to produce a normal trabecular structure. However, by comparison with eyes of approximately the same fetal stage in which obviously excessive numbers of cells are seen, the picture presented in Figure 9*A* is judged to be a normal one.

Figure 9*B* is an oil-immersion view of the chamber angle of a 220 mm. fetus. It shows the corneoscleral meshwork well along in development at *a*. The cells of the uveal meshwork are actively laying down collagen at this stage. The newly produced collagen takes a deep-amber to brown color in the Held stain, unlike either Descemet's membrane or older collagen fibers. A section through three of these new fibers arranged in a spiral manner, indicating a rudimentary minimal anterior border-ring of Schwalbe, is seen at *b*. Other uveal fibers are developing at *c*; the beginning of the chamber angle cleavage is seen at *d*, and cells of the ciliary body and iris root are at *e*.

The general impression gained is that the cells of the uveal trabeculae nearest the edge of the endothelium of Descemet's membrane differentiate and become active a little sooner than those nearer the scleral spur. As can be seen in Figure 9*A*, the largest nuclei of the uveal trabecular layers are at *a*. The dark-staining new collagen appears first in this position, sometimes as early as in 150 mm. embryos. This material is not easily identified in sections stained with hematoxylin and eosin, but it is a clear blue color with the Mallory stain, and amber to red with the Held and Held-Van Gieson stains.

Cavitation and Aqueous Formation

The opening of the chamber angle is an example of increasing volume by cavitation. Unequal growth of surrounding tissue and cleavage have been shown to be major factors in this action. A somewhat similar, but less obvious, increase in volume takes place within the trabecular meshwork, between the fibers. Apparently, the cells in this organ are so constituted that they do not adhere to adjacent cells along their sides if they differentiate as required for normal development. They adhere only at the tips of the collagen fibers in order to form long, smooth chains or stellate unions of several fibers. The fibers are then free to be displaced independently of the adjacent fibers, and measurable space can be found between them. This is partly due to the fact that the trabecular cells extend their increasing mass lengthwise during growth. Also, the corneal and scleral cells, at either end of the insertion of the trabecular fibers, grow much thicker and tend to wedge the trabecular fibers apart. Similarly, the normal proliferation and cell growth in the ciliary body wedge the uveal trabeculae apart at their uveal end.

If the development is normal in cornea, sclera, and uvea, the trabecular fibers are kept somewhat taut, contributing to the maintenance of space between the fiber layers. As the uvea changes shape, the layers of cells making up the meshwork are free to shift or slide. Thus, as the trabecular region, the scleral spur, the meridional ciliary muscle fiber insertion, etc., grow outward and forward around the iris root, it is possible for the inner uveal trabecular fibers to continue to elongate, maintaining their insertions in the ciliary body and iris root, which are not growing in the same direction.

As the spaces discussed are beginning to develop, and in order to make and keep them patent, they must be filled. The first production of aqueous has apparently been timed so that the fluid may serve this purpose. The coincident growth of the ciliary processes supports this theory. Just to balance the fluid volume against the cavitation may be all that is necessary, but it is conceivable that the aqueous pressure contributes dynamically to the cavitation.

The posterior and anterior chambers constitute, in a sense, a unit in which the pressure, as far as our present interest is concerned, is the same. The mild ballooning or filling effect of the fluid pressure within this unit creates adequate space within which normal growth processes can take place.

Perhaps in this way synechia formation is prevented.

Possibly, the newly formed aqueous helps to open the angle by actively "wedging" the tissues apart. However, this action cannot be limited to the anterior chamber. There is at least as much pressure behind the root of the iris as in front; so the "ballooning" or "wedging" must also help to move lens, lens zonule, and ciliary processes backward. Our feeling is that the aqueous pressure is normally little more than enough to fill the growing cavities, and therefore the importance of properly balanced fluid tension must be emphasized.

PERSISTENT FETAL ADHERENCE AS A CONTRIBUTING FACTOR IN DEVELOPMENTAL GLAUCOMA

Fetal chamber angles in which the uveal tissue is adherent to the trabecular zone have been seen by Shaffer, Maumenee, and Scheie,‡ as well as by others (see the literature cited by Anderson[3]). Figure 10*A* shows the chamber angle of a fetus from the collection of Shaffer and Maumenee, reproduced here, with their permission.§ A prominent ring of Schwalbe is seen at the anterior edge of the still actively growing trabecular meshwork. The trabecular mass appears to be torn away from the cornea approximately in the area where Schlemm's canal should be found. Schlemm's canal cannot be identified. In spite of the age of the fetus, the ciliary body does not appear to be well developed. This may contribute to the adherence of the peripheral uveal tissues to the trabecular tissue. The adherence covers approximately the posterior half of the meshwork. The adherence might have persisted to the adult stage, so that the chamber angle might then have presented the picture so commonly misinterpreted as embodying "mesodermal remnants."

‡ Shaffer, R. N.; Maumenee, A. E., and Scheie, H.: Personal communications to the authors.

§ No information about the age of this fetus is available. It is our impression that its age is approximately 8½ fetal months, and Dr. Shaffer concurs in this opinion.

The chamber angle of the congenitally glaucomatous eye of Patient M. M., aged 18 years, enucleated after trauma, shows persistent adherence of the posterior half of the meshwork (Fig. 10*B*). Other typical developmental features are also seen. The trabeculae are poorly formed, lacking the normal geometric structural appearance. The collagen, the elastic fibers, Descemet's membrane, and endothelium appear not to be organized, so that they make up an irregularly interwoven form. In other words, the collagen is not always surrounded with any or all of its covering layers, nor are the layers found in an organized relationship to each other.

Perhaps partly as a result of this diffuseness of elements, the inner uveal fibers are about one-third their normal length. Associated with this, the circular ciliary muscle bundles are situated far forward. Schlemm's canal is absent.

The base of the uveal meshwork is fanned into abnormal width, but, while this may seem to indicate excess material, it is not in the nature of the so-called mesodermal remnants. First, if the material within this area could have differentiated normally and could have been properly distributed, some of it might have gone into tripling the length of the innermost trabecular layer. It would then probably be seen that no excess existed. Second, the material is within the trabecular mass, and not in the apex of the chamber angle.

The other eye of the patient was found to have an increased intraocular pressure. A trephining controlled the pressure, and a gonioscopic drawing (Fig. 10*C*) was made. The very thin iris was seen to be attached near the anterior border of the trabecular tissue. Some ciliary processes were incarcerated in the trephine opening. The ciliary processes (*a*) which were not incarcerated seemed to rest far forward against the posterior iris surface. The first eye was not seen gonioscopically, but undoubtedly gonioscopy of that eye would have revealed much the same appearance as that in the second eye, except for the surgical coloboma.

Another eye in our collection, from a patient with infantile glaucoma (M. T., aged 5 years) showed, aside from a secondary membrane across the angle, the same anomalies as did the eyes of the previous patient. Here, again, Schlemm's canal could not be identified in any section, the region being completely filled with sclerotic tissue. The uveal trabeculae were about one-half their normal length and lacked somewhat in organization.

From these and other examples we concluded that a salient feature of developmental glaucoma was a failure of the normal development of the trabecular system, through which opening of the chamber angle by cleavage was incomplete or absent. This abnormality may result from a failure of cellular differentiation or from a failure of aqueous to form at the proper time or from a combination of the two.

The favorable effects of goniotomy can be ascribed to a breaking through the persistent fetal adherence of the chamber angle structures or through abnormal trabecular tissue or, in some cases, both, with corresponding increase in aqueous drainage. The result would thus be the same as though hypothetical mesodermal remnants had been cut through.

PECTINATE LIGAMENT ELEMENTS AND PROMINENT ANTERIOR BORDER-RINGS OF SCHWALBE

Fibers in the chamber angle, reminiscent of the pectinate ligaments, and the pearly-white, prominent anterior border-rings of Schwalbe have both been described long ago.‖ The predisposition to glaucoma of patients with moderate to extensive pectinate ligament anomalies has been well established. However, the manner in which these structures come about, their interrelationship, and their relationship to the glaucomas have not been made entirely clear.

These tissues are obviously excessive to the anatomic and physiologic requirements of the normal human eye. Because of this, and the common acceptance of the atrophy theory of chamber angle development, even these organized architectural features have been classed as mesodermal remnants by several authors.¶ Again, the implication is that the rudimentary tissues are present in most embryos, and that in the normal course of events the undesirable mesoderm degenerates. If this were true, we should be able to identify the excessive tissue in almost every embryonic or fetal eye at some time earlier than the beginning of the fifth month. In our collection, consisting of 19 eyes from embryos ranging from 11 to 220 mm., 12 were from embryos under the 100 mm. stage. Among these 14 eyes, there were found only 3 showing cell groups which might be considered to represent such excessive tissue. One eye, of a 91 mm. embryo, which will be discussed in a succeeding paper, contained a very prominent mass of tissue, while there were only slight traces in the other eye. In the former, the cells had differentiated and were actively producing collagen which stained like the heavy fibers found in the adult. Our study of such collagen in pathologic adult eyes indicates that such material resists atrophy. The impression gained was that these cell groups would continue to differentiate and that a strong and permanent structure would result.

The adult form of these anomalies were described in the preceding paper, with their histologic, external slit-lamp, and gonioscopic appearances contrasted and related to the normal. Embryonic and comparative anatomical studies give us a reasonably sound explanation of their source and course of development. These latter are reported in a succeeding paper.[2]

SUMMARY

The angle of the human anterior chamber opens during fetal development by a process of cleavage, a simple separation between two mesodermal layers, in a manner slightly similar to the opening of the anterior chamber proper during a much earlier stage of embryonic life.

‖ See the literature cited in Reference 1.

¶ See the literature cited in Reference 1.

The forces needed for this cleavage are supplied by the very extensive unequal fetal growth of the chamber angle tissues, resulting in changed spatial relationships.

No process of atrophy and resorption of mesodermal tissue is required, and none takes place in measurable amount.

Abnormal variations of the human chamber angle, such as prominent rings of Schwalbe and pectinate ligament features, should not be thought of as "mesodermal remnants." They are the result of abnormal or excessive laying down of cells.

The pathogenesis of certain cases of developmental glaucoma can, at least in part, be traced to poor differentiation and organization of the cells within the trabecular structures and to persistence of fetal adherence of mesodermal structures, i. e., to a failure of the chamber angle to open fully by cleavage. In these cases, at least, the favorable results of goniotomy must be attributed to a severance of this adhesion or of abnormally developed trabecular tissue, rather than to a stripping or cutting of nonexisting "mesodermal remnants."

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The Anterior Border Ring of Schwalbe and the Pectinate Ligament

Embryologic and Comparative Anatomic Studies

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EMBRYOLOGY

In a previous paper [1] it was shown that no tissue requiring atrophy and resorption is present in the typical chamber angle of the embryonic or fetal eye. A comparison of cell layer counts in fetal and adult eyes showed that there is on the average an increase in the number of cell layers between Schlemm's canal and the pigment epithelium of the ciliary body during the period of cleavage of the angle. The increase appears to be entirely within the ciliary-iris tissue. By contrast, the increase in size of the mass of trabecular tissue and the ring of Schwalbe seems to be due to cell growth and cavitation.

To say that no tissue requiring atrophy is present in the average fetal angle does not mean that excessive tissue is not present in a certain percentage of cases. Excessive embryonic mesoderm is seen, but only approximately in the same percentages in which similar, but fully developed, tissue is found in adult eyes. It is also noted that in some embryos collagen has been produced in these layers long before cleavage begins. Collagen in the same positions in adult eyes has been seen to persist through pathological changes which have nearly obliterated other cell elements. This seems to be a further indication that once such material is present in the embryo or fetus, it will normally change only in the direction of further development.

Many fetal chamber angles of all ages contain no excessive cells along the line at the periphery of Descemet's membrane and the anterior border of the trabecular tissue or along the inner surface of the latter. Others contain a very few extra cells, which might be expected to develop into mildly prominent rings of Schwalbe and pectinate strands. Sections of such fetal angles, as well as of fetal angles showing a large bundle of cells already formed into a prominent anterior border-ring of Schwalbe, were pictured in the paper mentioned.[1]

Figure 1 of this paper is an oil-immersion view of the angle of a 220 mm. fetus. The cleavage along the inner surface of the trabecular tissue is already taking place; a few irregularly aligned cells are still adherent at the fetal angle, and progression of the schism peripherally can be seen. Collagen-like material being laid down at *a* gives the impression that a single row of cells around the angle may tend to lead to the formation of a ring of Schwalbe and pectinate strands. However, by comparison with other fetal eyes, it is probable that this attachment is too weak to persist in the face of otherwise normal development. Probably the faintest pearly line would be visible gonioscopically in the adult eye.

The cell at *b* in this Figure is of the iris or ciliary uvea and will lie on the posterior side of the chamber angle cleft, while cells *c* and *d* are part of the uveal trabeculae. The arrows at *c* and *d* point to the newly formed collagen, which in the Held stain takes a

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deep-amber coloration. The collagen at *c* can be traced forward to the nucleus of its cell, and it can be seen attaching to the collagen of another cell. The material at *e* is similar to the trabecular collagen, but will become part of the pattern of fibers of the iris.

An early appearance of a prominent anterior border-ring of Schwalbe is shown in histologic sections of the chamber angle of a 91 mm. fetus (Fig. 2). The clump of extra cells seen at *Schw.* (Fig. 2*A*) is a cross section of a ring-like prominence which encompasses almost the entire chamber angle. In many places, the ring has been torn artificially from the corneal side of the chamber, so that it appears to belong to the iris root (Fig. 2*B* and *C*), but in many places the torn condition of the cells indicates the original relationship of this ring to the trabecular tissue. In the sections stained by the Mallory method, a collagen element is clearly visible between the cell nuclei within this group, and in the nearly flat sections, to the sides of the eye (Fig. 2*D*), masonry-like courses of blue-staining fibers (*Schw.*) demonstrate the already well-developed circular fibers. In this section, again, the artificial opening of the chamber is evident, and a more natural, clear-cut, though narrow, cleavage line is seen at *Cl*.

It is anticipated that in a larger embryologic collection than we have at our disposal all the stages of development of the anterior border-ring of Schwalbe could be found.

We believe that certain cells of the rudimentary anterior border-ring of Schwalbe are so oriented as to interlock with cells at the anterior tip of the inner uveal trabecular group, as at *r* in Figure 2*A*, and that these become firmly adherent after adequate development of the collagen of the latter. If certain special cells, destined to form the pectinate ligament, are laid down peripherally to the rudimentary ring of Schwalbe, such as are present as *p* in Figure 2*C*, these will develop in a plane with the ring, in a layer separate from both the trabecular group, peripherally, and the iris-ciliary tissue, centrally. A barely perceptible cleavage line can be seen extending from the anterior chamber between Schwalbe's ring and the iris-ciliary tissue, going to *a* in Figure 2*C*. In later stages this cleavage line should develop into a cleft or opening. In these later stages another cleavage line should also appear between the trabeculae and the pectinate strand

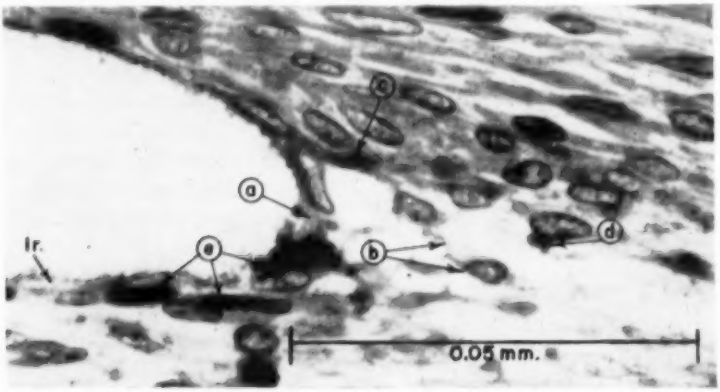

Fig. 1.—Section (Held stain) through the chamber angle of a 220 mm. fetus: (*a*) amber-staining collagen being laid down by the cell oriented across the apex of the fetal angle; (*b*) nucleus and collagen of an iris cell temporarily adherent across the developing cleft; (*c*) dark-amber-staining tip of the collagen fiber being laid down by a uveal trabecular cell; (*d*) cross section of dark-amber-staining collagen of a longitudinally oriented uveal trabecular fiber; (*e*) collagen of iris cells.

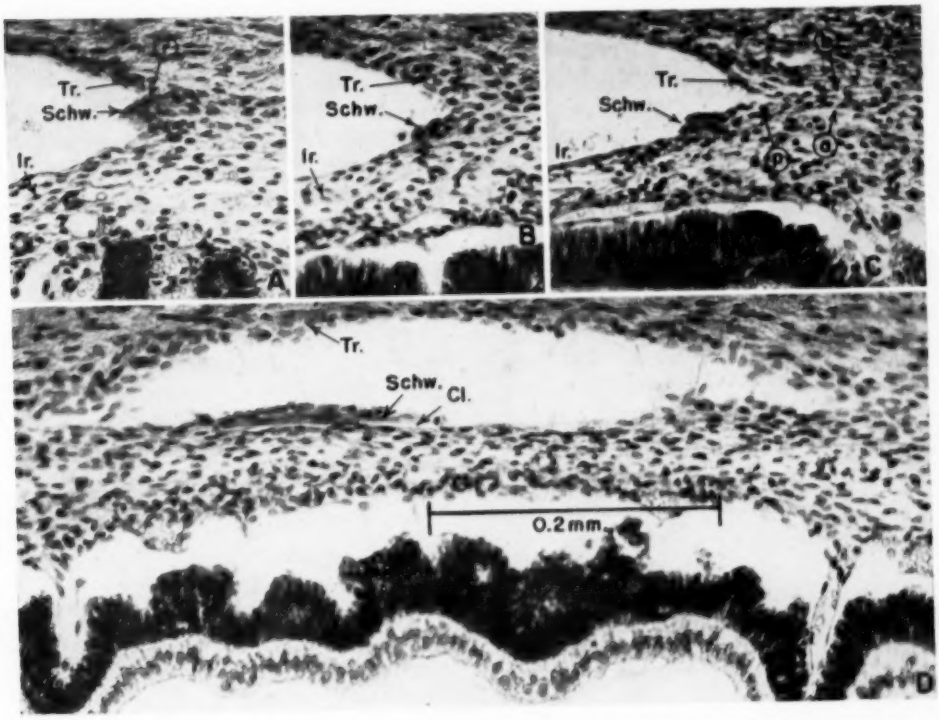

Fig. 2.—Sections (Mallory stain) through the chamber angle of a 91 mm. fetus.

A: Schw., cells, excessive for the typical human chamber angle, constituting a rudimentary stage of a prominent anterior border-ring of Schwalbe; *r*, point at which the ring of Schwalbe unites with the middle layers of trabecular fibers; *Tr.*, trabecular cells anterior to the ring of Schwalbe and the eventual periphery of Descemet's membrane; *Ir.*, iris mesoderm.

B: the ring of Schwalbe artificially detached from the trabecular tissue and resting against the root of the iris.

C: p, probably a rudimentary pectinate ligament strand; *a*, potential cleft which will separate the strand from the iris-ciliary body tissue; *b*, potential cleft which might separate the strand from the trabecular tissue.

D: nearly tangential section of the eye showing a longitudinal section through the anterior border-ring of Schwalbe, which is artificially detached from the trabecular tissue. *Cl.*, a clear line revealing the true potential plane of cleavage between the ring of Schwalbe and the iris-ciliary tissue.

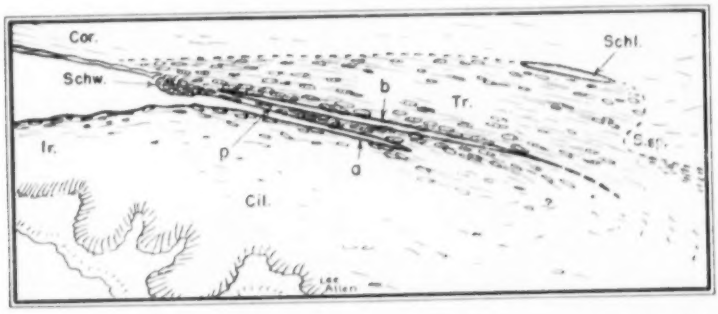

Fig. 3.—Schematic drawing showing the two planes of cleavage, *a* and *b*, which isolate a pectinate strand (*p*) from the iris-ciliary body tissue and the trabecular tissue, respectively.

(as indicated at *b* in Fig. 2*C*). This latter cleft would stop short of the anterior border-ring of Schwalbe, anteriorly, and end in the region of the scleral spur, peripherally. The two cleavage lines are represented diagrammatically in Figure 3.

The material in the pectinate ligament is not a solid sheet, but is arranged in separate strands, so that there are spaces between the individual pectinate strands. Therefore, clefts *a* and *b* in Figure 3 actually are joined, and aqueous may flow freely between them, much as in the animal eyes aqueous flows freely around the pectinate fibers and through the maze of the spaces of Fontana.

In the animal eye, the anterior border-ring of Schwalbe and the pectinate ligament are parts of the same suspensory system of the iris root (Troncoso[2]). In man they appear to be genetically determined throwbacks. Therefore, in man this suspensory system does not often appear as a full-blown, well-organized structure. Parts only, or we might say fragments, of the whole structure may appear. The prominent ring may be the only element present; it is seen in about 15% of all human eyes. The peripheral rows of pectinate fibers may appear without a prominent anterior border-ring of Schwalbe, but frequently a pearly-white line is associated with them along some extent of the chamber angle. Lastly, there are those eyes in which at least a few of the pectinate strands unquestionably insert into a more or less prominent border-ring of Schwalbe, in exactly the same manner as in lower animals.

Several investigators, Troncoso[2] and others, have already concluded that these anomalous uveal forms are identical with parts of the pectinate ligament of lower animals. They have assumed that in animals these tissues go on to full development, thus satisfying the functional needs in the respective eyes. Therefore, the broad impression left by a survey of the relevant literature has been that rudimentary mesoderm for the formation of these structures is present in most human embryos and that it atrophies in the course of development because it is no longer needed.

We agree with the earlier authors in believing that the anomalous uveal structures are parts of the pectinate ligament complex and therefore represent atavistic formations in the chamber angle, but we do not believe that they are regular features in the development of the human which are expected to atrophy in the ordinary course of events, and persist into the adult eye in exceptional cases. Out of 19 embryonic and fetal eyes, 3 showed some rudimentary rings of Schwalbe, and 1 of these to a marked degree. These percentages are approximately the same as those referring to incidence of prominent rings of Schwalbe in adult eyes.

COMPARATIVE ANATOMY

To gain additional material for the comparison of the chamber angle in man and that of other mammals, we repeated parts of the study so well conceived and described by Troncoso.[2] Our preparations were of the anterior chambers of bovine, pig, rabbit, and dog eyes. We wished to discover the variations, as well as similarities, in the tissues at the iridociliocorneoscleral angle among the individuals, as well as the species. It was hoped that the findings might help to explain the varied appearances of similar tissues in the human chamber angle.

Space does not allow a comprehensive report of the study, and only a few of the pertinent details can be mentioned in support of the broader conclusions.

We found that we could reevaluate some conclusions of others regarding the nature of the tissues at the chamber angle in both the animals and man. As an isolated example, the simple statement by some authors that the pectinate ligament inserts anteriorly at the end of Descemet's membrane must be qualified. Only the row of pectinate strands nearest the pupil appears always to insert at that line. Many of the more peripheral strands bridge to the trabecular tissue, where some appear to unite with trabeculae well behind the ring of Schwalbe, while others

rest against the trabecular tissue in the remainder of their course up to the point at which they insert into the ring of Schwalbe. This is of importance in the morphology of prominent rings of Schwalbe and pectinate ligament strands in the human eye.

An attempt is made to picture the most important similarities and differences between the lower mammalian chamber angles studied and the human in the semischematic drawings (Fig. 4*A* and *B*). Figure 4*A* is a compilation of features found in four animals, with the general contours based largely upon those of the pig. Figure 4*B* represents the human chamber angle with a prominent ring of Schwalbe and a pectinate ligament strand moderately isolated from the trabeculae, and therefore bridging the angle. Similar structures in the two drawings bear the same labels.

At the external wall of the scleral groove we find Schlemm's canal (*Schl.*), in both man and animal.* The structures remaining between Schlemm's canal and the anterior chamber are identified by arabic numerals.

1. The most external layers of trabecular fibers are adjacent to Schlemm's canal. A number of these rest entirely within the scleral groove and relate only to corneal and scleral tissue. At their anterior ends they insert into the scleral septum or the corneal stroma or both. Posteriorly the fibers insert into the scleral spur.

2. The next, more internal group of fiber layers is somewhat variable, but with extensive and careful study it can be shown that it relates to the uvea. Some of the fibers at the anterior insertion pass into the posterior

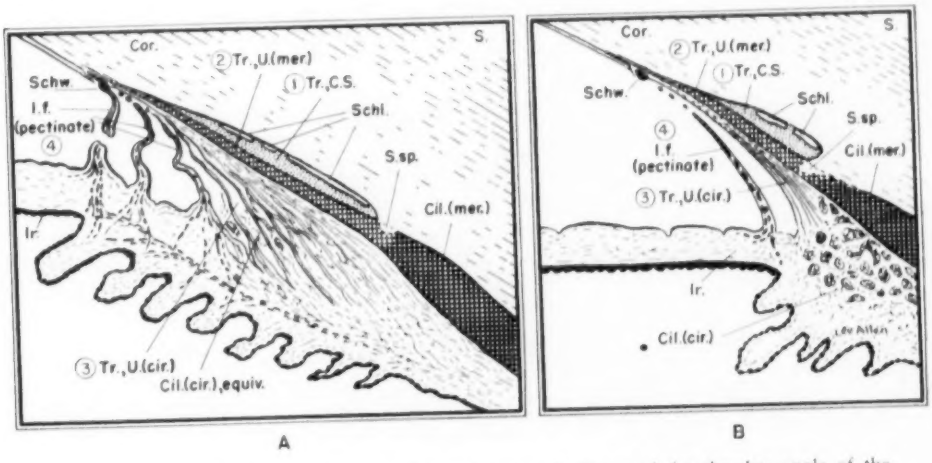

Fig. 4.—*A*, schematic drawing, based largely upon the forms of the chamber angle of the pig, but combining some features of the angles of other lower mammals, showing the four major groups of fibers lying between the anterior chamber and Schlemm's canal (*Schl.*). *1 Tr., C.S.*, corneoscleral trabecular fiber group; *2 Tr., U. (mer.)*, uveal trabecular fiber group associated with the meridional ciliary muscle; *3 Tr., U. (cir.)*, uveal trabecular fiber group associated with the radial and circular ciliary muscle bundles; *cil. (cir.), equiv.*, the region which is equivalent to the region in man containing the radial and circular ciliary muscle bundles; *4 If. (pectinate)*, iris fibers or pectinate strands; *Schw.*, anterior border-ring of Schwalbe.

B, schematic drawing of one type of human chamber angle, showing the same four structures lying between the anterior chamber and Schlemm's canal as found in the lower animal. The figures clearly demonstrate the differences between *A* and *B* in proportion and position of the various elements.

* In spite of the claims of some authors that a true canal of Schlemm exists only in primates, we find no reason to think that the channels situated in the scleral groove in the animal do not function exactly as in man. We agree on this point with the observations of Anderson,[3] Walls,[4] and others.

layers of the corneal stroma, while in some eyes a few fibers are incorporated into prominent rings of Schwalbe. The posterior insertion of this group is always into the meridional ciliary muscle, either directly or indirectly. This relationship is undeniable in some individual animal eyes, where most of the fiber layers pass internal to the tip of the sclera spur and only one or two layers interweave between circular fibers of the tip of the spur to reach their insertion in the external layers of the meridional ciliary muscle fibers. This group, in man, becomes more involved with the scleral spur, but in a few persons more than half the layers pass internal to the spur. In preparations where the ciliary muscle, some of the trabecular fibers, and Descemet's membrane have been torn away from the corneoscleral wall, the tip of the scleral spur is always torn away with the former. Occasionally fibrils can be traced from the trabeculae, between the cut sections of the circular scleral fibers, and can be seen to insert into the meridional muscle. The fibers of this group take a very direct meridional course, and often the entire length of a fiber can be seen in histologic sections.

3. The third group of fiber layers has its posterior insertion in the connective tissue around the radial and circular ciliary muscle bundles. In some sections of some eyes, a few of these uveal trabeculae seem to relate to the walls of vessels, such as the major circle. The anterior insertion of the group is at the edge of Descemet's membrane. At this level, if there is a prominent ring of Schwalbe, the fibers take a more circular course and blend with the ring. In those eyes where the Schwalbe line is not prominent, but represents only an anatomical limit, this third group of trabecular fiber layers thins out to one or two layers and is recognized in histologic sections as a group of somewhat separated circular fibers. Often, some of the fibers take a completely circular direction short of the edge of Descemet's membrane and never reach that level. In rare instances, as in Figure 5, the anterior limit of this third group is displaced backward in the human eyes so that it does not reach the edge of Descemet's membrane at all. This was noted in 3 eyes of the 600 studied histologically, and has been suspected several times in gonioscopic examinations of living eyes. There is a question as to how the term "anterior border-ring of Schwalbe" should be applied in such cases, since most of the material within prominent rings of Schwalbe is related to, or possibly made up of, extensions of the internal uveal trabeculae. Perhaps the term should be broken down, "Schwalbe's line" still being applied to the boundary between the trabeculae and Des-

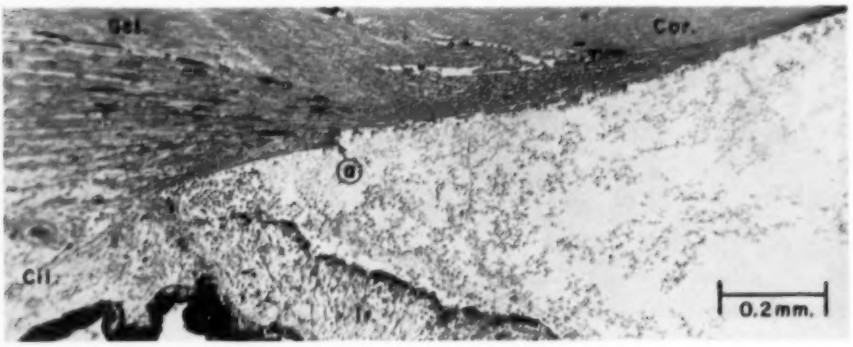

Fig. 5.—Section (Held stain) through the chamber angle O.D. of Patient M. S., a man aged 21: (*a*) exceptional posterior position of anterior insertion of the inner uveal trabecular fibers (Group 3 of those shown in Figure 4*B*).

cemet's membrane, while "ring of Schwalbe" would be applied to the circular fibers usually found at that level.

4. The most internal structure in the animal eyes is the pectinate ligament. It may be made up of several rows of strong uveal strands originating in or near the root of the iris and terminating on the corneal side of the anterior chamber at the end of Descemet's membrane. The spaces between these strands have been called spaces of Fontana and have been assumed to play a major part in the elimination of aqueous through the uveal vascular system.

Man normally has no pectinate ligament, supposedly because his physiologic requirements differ from those of most animals. It was one of the purposes of this investigation to find out whether the anomalous uveal strands found in many human eyes do have any real anatomic relationship to the pectinate ligament of animals, as Troncoso[2] believed.

The most central row of pectinate strands is usually made up of thick bundles of cells, rather than single fibers, as are the trabeculae. The successively more peripheral rows are usually made up of thinner strands, tending to look more and more like trabeculae. As stated earlier, the most central strands insert at the end of Descemet's membrane. In some eyes, such as our bovine specimens, these strands take on a nearly circular direction as they approach the cornea; some run along the inner surface of Descemet's membrane for a way before penetrating or passing around its edge to blend with some collagen of the inner uveal trabeculae. In other eyes, such as in one rabbit's eyes, the strands bridge and insert in a more directly meridional manner, and the insertion is just barely around the edge of Descemet's membrane and slightly onto its anterior surface. The more peripheral strands in any of the eyes may insert anywhere along the inner surface of the uveal trabeculae or pass along the surface to insert into the ring of Schwalbe.

The collagen core of the pectinate strands always takes a deep stain at their insertions, and this stain is identical with that in the prominent rings of Schwalbe in man. It is always different from that taken by Descemet's membrane.

The posterior insertion of the pectinate ligament is variable. Many of the strands send collagen fibrils far into the ciliary body. However, some of the most central strands seem to relate to the stroma of the iris root. This is especially true in some of our specimens, such as the dog eyes, where many of the strands blend gradually into the iris.

In the human chamber angle, as shown in Figure 4*B*, the pectinate ligament strands are not usually so regularly arranged. Often, in an eye, only one or a few scattered "fractions" are found. What has been said of the make-up of the strands and their variable anterior insertions can be applied to the human. However, in our human specimens the posterior insertion is into the stroma of the iris only. Even where the bases of the pectinate strands are directly over the circular ciliary muscle, no fibrils have been seen extending into the latter organ. This is no indication that these strands are not in the nature of pectinate ligament features, since we find similar posterior insertions in some animal eyes.

When the four tissues just described are compared as to general character and distribution, certain facts become evident.

1. It is true that some of the fiber layer groups described above generally have a differently detailed structure; some authors described certain of these layers in the animals merely as spongy tissue. In certain species, a layer which appears "spongy" in one animal's eye will in another animal's eye take on the appearance of well-organized fibers, similar to those in the human.

2. We found, as did Anderson,[3] in both man and animal, that the inner uveal trabeculae (Group 3, Fig. 4) very seldom contain any elastic fibrils, while those external to this group almost always contain elastic elements.

The presence of elastic fibrils, not pictured here, within many prominent rings of

Schwalbe in man, even though most of the uveal fibers seen to enter are from the inner uveal group (*3*, Fig. 4), is further evidence that fibers of the outer uveal group (*2*, Fig. 4) are also incorporated in its mass.

3. When the four main tissues described above are pictured, as in Figure 4, it can be realized that there is more similarity between man and some mammals below primates than has been appreciated by some relatively recent investigators. Aside from the obvious fact that the pectinate ligament elements are not present in the average human eye, the greatest difference seems to be that all of the fiber layers between the anterior chamber and Schlemm's canal are more compactly arranged near the corneoscleral coating of the eye in man than in animals.

Our comparative anatomic studies have helped to elucidate the meaning of pectinate ligament strands and prominent anterior border-rings of Schwalbe in the human chamber angle. Because we and others have noted the familial character of these anomalies in man, and because they bear such similarity to corresponding structures in other mammals, their presence is judged to be the result of more or less exaggerated, genetically determined atavistic traits. Recognition of the origin of these anomalies adds weight to the objections against the theory that atrophy and resorption of mesodermal structures occurs normally during the development of the human chamber angle in order to remove such mesodermal tissue as might prevent normal formation and proper functioning of the filtration angle.

SUMMARY

Prominent anterior border-rings of Schwalbe and pectinate ligament features in man are the result of excessive laying down of mesodermal tissue in embryonic and fetal eyes. They do not result from a failure of atrophy and resorption of mesodermal tissue normally laid down in each embryo.

Comparative anatomic studies show that analogies to all forms of pectinate fiber anomalies occurring in man are found in the normal chamber angles of animals. In man they represent genetically determined atavistic traits.

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Rolling of the Eye Around Its Visual Axis During Normal Ocular Movements

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DEFINITIONS

In European literature rotation of the eye around its visual axis is usually called *rolling*. *Apparent rolling* during rotation of the normal eye around an oblique axis in Listing's plane is called *torsion*. In American literature rotation around the visual axis is usually called *torsion* and apparent rolling during rotation of the normal eye around an oblique axis in Listing's plane is called *false torsion*. It would eliminate confusion if one or the other group would find acceptance.

In this paper the following definitions will be used:

1. Rolling—rotation of the eye around its visual axis.
2. Torsion (or false torsion)—apparent rolling when the normal eye rotates about an oblique axis in Listing's plane.

COORDINATE SYSTEMS

A coordinate system is anything which is used as a reference to locate the position of a point in space. It could be the horizon, a series of horizontal or vertical lines or planes, the spherical system used here, or anything else. The only criterion is that it should be a useful standard for determining the position of an object in space.

In the planar coordinate system the position of a point is determined by its relation to horizontal and vertical lines and planes, the position of which is already known. This system is, of course, widely used, but when it is employed in observations of the surface markings on a rotating sphere serious errors are introduced. These errors appear as "torsion" or "false torsion." When the eye is rotating around an oblique axis in Listing's plane, torsion is seen as the angle that a vertical plane, passing through the center of rotation of the eye and the fixation point, forms with the meridian of the cornea or retina which was vertical when the eye was in the primary position. Unfortunately, rolling, if present, manifests itself in exactly the same way. When the planar or rectangular coordinate system is used, the observer sees the algebraic sum of torsion and rolling, and is unable to distinguish between the two. No such errors occur when the spherical coordinate system is used.

A spherical coordinate system is made up of lines of longitude and latitude, like those on the surface of a globe map of the earth. The lines of longitude are great circles produced where flat planes passing through both poles and intersecting in the polar axis cut the surface of the sphere. Flat planes perpendicular to the polar axis cut the surface in the lines of latitude.

It was shown by Putnam[1] that to avoid false hyperphoria when the movements of both eyes are to be observed in measuring squint, the polar axis of the spherical coordinate system should lie horizontally in Listing's plane and pass transversely through the center of rotation of the eye (Fig. 1*A*). Each eye should have its own separate coordinate system arranged in this manner.

When one is studying rolling and torsion, the spherical coordinate system should be used differently. Its polar axis should run

From the Department of Ophthalmology, Reading Hospital.

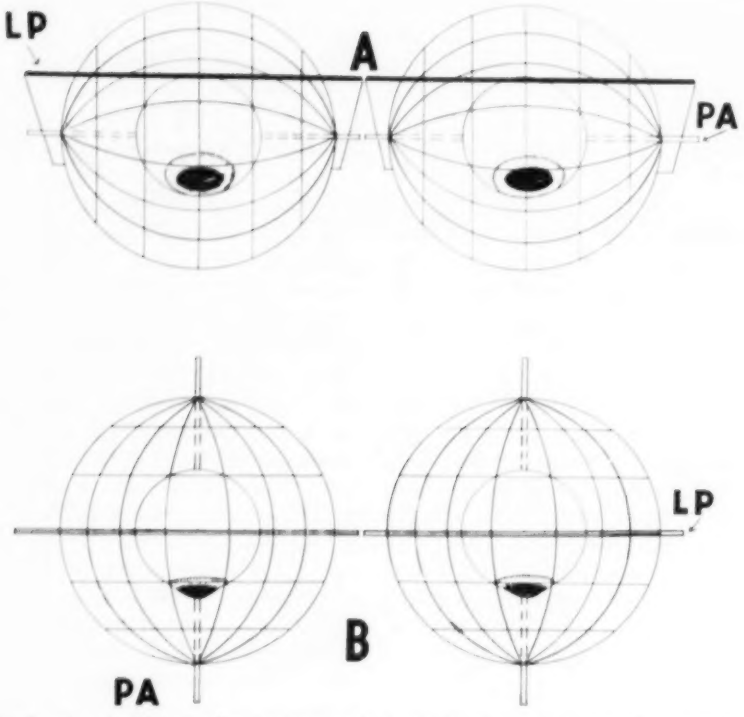

Fig. 1.—*A*, spherical coordinate system as it should be employed in measuring squint. *PA*, polar axis; *LP*, Listing's plane. The coordinate system does not move with the eye.

B, spherical coordinate system employed to measure rolling. Polar axis is fixed in primary position.

in an anteroposterior direction, passing through the center of rotation of the eye and the primary position (Fig. 1*B*). The equator of the coordinate system should lie in Listing's plane. When one is looking for rolling, the patient's eye should be made to rotate so that the visual line follows one of the lines of longitude toward or away from the primary position. The meridians of the cornea and retina will then maintain a constant angle with the meridian of longitude which the visual line is following. Any change in this angle will be due to rolling. Torsion will not complicate the observation.

PRESENT STUDY

To study rolling during normal rotations of the eye in relation to this spherical coordinate system, a transparent plastic hemisphere (Fig. 2) was mounted on a support with a headrest and chin rest eccentrically placed so that the subject's right eye would be at the center of the hemisphere. (The hollow column of the headrest fits over either of two wooden pegs to bring either the right or the left eye to the correct position for testing.) Strips of plastic tape were placed on the surface of the hemisphere to represent the meridians of a spherical coordinate system, as in Figure 1*B*. At intervals along these spherical coordinates, fixation points were marked with a wax pencil on the concave surface of the sphere, as indicated by the position of the round black dots on the convex surface. As the subject looked in turn at each of the fixation points, the position of her blind spot, indicated by black squares, was plotted with a 10 mm. target (at 265 mm.). Allowance of 2 degrees was made for the slightly inferior position of the blind spot on the retinal horizon. The white adhesive strips connecting the blind spots and fixation points show the projected positions of the retinal horizon. Eight subjects were tested, the youngest 11 years of age. The results were so nearly identical that the white strips are used to represent the average positions of the retinal horizons of all eight. To avoid confusion, only the averages for the right eye are illustrated in the Figure.

RESULTS

It is shown by the white strips that when the eyes moved vertically or horizontally from the primary position there was no rolling. At first glance it would seem that there was a large amount of rolling as the eye rotated outward from the primary position along the oblique meridians *B* and *D*, but the measured angle between those plastic strips, representing the retinal horizon, and the oblique meridians of longitude remains the same as when the eye was in the primary position; therefore there was no rolling. It is only because the observer uses horizontal and vertical lines for reference that this angle seems to increase. This apparent increase is torsion (or false torsion). Only that meridian of longitude which the visual line is following may be used for reference to determine rolling.

ROLLING

When the eyes moved obliquely far out into the superior and inferior temporal quadrants of the field of fixation at *B* and *D* (Fig. 2), there was an increase of 3 to 5 degrees in the angle between the retinal horizon and the 45-degree meridian of longitude the eye was following (measured with a protractor). There was, therefore, slight outward rolling when the eyes moved obliquely upward and outward, and inward rolling when the eyes moved obliquely downward and outward. Adler * predicted that such outward and inward rolling would be found in the upper and lower oblique directions in the normal eye. The present study proves this to be correct. Corresponding rolling could not be demonstrated in the smaller, nasal field of fixation. This rolling in extreme degrees of oblique rotation has nothing to do with torsion.

ROLLING IN MOVEMENTS OTHER THAN THOSE TOWARD OR AWAY FROM THE PRIMARY POSITION

If the visual line which has been following a given meridian moves to a new meridian which has a different direction in relation

Fig. 2.—Projection of retinal horizon for various directions of gaze. Long plastic strips on transparent hemisphere represent meridians of longitude or spherical coordinate system. Polar axis passes through center of rotation of eye and primary position. Round black dots show fixation points on meridians of longitude; black squares, positions of blind spot; short white strips, projections of retinal horizon. Points *A*, *B*, *C*, and *D* are limits of field of fixation. White strips beyond *A*, *B*, *C*, and *D* are hypothetical and only illustrate nature of compensatory rolling. Test target used is almost as large as blind spot.

to the primary position, the eye undergoes rolling as it moves to the new meridian. The retinal horizon and this new meridian of longitude assume the same angle they would form if the eye were in the primary position. This is illustrated by the results of the present study. As the visual line leaves the vertical meridian at *A* (Fig. 2) and travels counterclockwise to the 45-degree oblique meridian of the upper temporal quadrant, at *B*, then to *C* on the horizontal meridian, and finally to *D*, the retinal horizon assumes with each meridian crossed the same angle it would form with this meridian at the primary position. In producing the amount of compensatory rolling necessary to cause the meridians of the cornea and retina to assume a "primary-position" angle with each new meridian of longitude the visual line crosses, the eye rotates around an axis lying in the secondary

* Adler, F. H., in discussion on Quereau.[2]

axis plane. This is a plane which bisects the angle formed by the intersection of Listing's plane and the plane of the equator of the eye. It lies halfway between the two. The strips beyond *A*, *B*, *C*, and *D* are outside the field of fixation and are intended only to illustrate the nature of this rolling. A line parallel to the uppermost plastic strip which represents the position of a hypothetical projection of the horizontal retinal meridian at this point would, if extended, be almost perpendicular to the horizontal meridian. The eye would have to roll clockwise around its visual axis about 90 degrees in order that the retinal horizon should be horizontal on arriving at meridian *C*. The position at meridian *B* is the midpoint in this clockwise rolling.

If the projection of the retinal horizon in the hypothetical position beyond point *A* were to move counterclockwise in a circle without rolling clockwise around the visual axis, it would cross each meridian at a right angle. The optic nerve and muscles would be twisted off the eyeball. Clockwise rolling around the visual axis counteracts this twisting. For a small circle around the primary position there is almost no necessity for compensatory rolling. The more nearly the size of the circle approaches the limits of the field of fixation, the more compensatory rolling is required. If the visual line makes a clockwise circle, the compensatory rolling is counterclockwise.

The plastic strips at points *A*, *B*, *C*, and *D*, which are the average result of testing eight subjects, shows that the eye in passing from meridian *A* to meridian *C* underwent about 45 degrees of clockwise, or inward rolling, as measured with a protractor. It appears that the retinal horizon first underwent outward rolling, then inward rolling, as the visual line moved from point *A* to *B* to *C*. Actually, if a line is drawn parallel to the plastic strip at point *A*, the line will meet point *B* at an angle greater than that formed by the plastic strip already there, showing that the eye made a clockwise movement of inward rolling in going from point *A* to point *B*. (These are spherical angles.)

COMMENT

A perimeter can be used to determine the position of the blind spot for various positions of gaze. Small pieces of tape at intervals on the inner surface of the perimeter arms serve as fixation points. Larger pieces of tape mark the positions of the blind spots. A perimeter embodies the spherical coordinate system illustrated in Figure 1*B*.

Use of the blind spot to detect pathological rolling is illustrated by the following case:

L. C., age 14, has had paresis of the left inferior oblique muscle of six years' duration, from an unknown cause. When both blind spots are plotted on a 1-meter tangent screen, the left blind spot is about 4 degrees higher than the right, showing inward rolling of the left eye consistent with paresis of the left inferior oblique.

SUMMARY AND CONCLUSIONS

Plotting the positions of the physiological blind spot for various directions of gaze is recommended for observation of rolling in normal and pathological rotations of the eye.

The positions of the blind spot show that in movements of the eye other than toward or away from the primary position, a meridian of the retina, by rolling around its visual axis, assumes the same angle with the meridian of longitude it is crossing that it would if the eye were in the primary position.

Slight normal outward rolling occurs when the eye is moved obliquely into the upper temporal quadrant, and inward rolling occurs when it is moved obliquely into the lower temporal quadrant.

Use of a spherical system of coordinates eliminates torsion and thereby gives a true picture of rolling.

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Antibody Formation in Rabbit Eye Studied with Fluorescein-Labeled Antibody

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Previous experimental work with Leptospira pomona infection in the rabbit eye indicates that antibody formation takes place locally and is related to the infiltration of the uveal tissue by plasma cells and lymphocytes. This concept is based on the observations that the antibody content of the gamma-globulin of the aqueous humor ("antibody activity") in local infection was higher than the activity of the serum and that the amount of local antibody activity was roughly correlated with the degree of plasma cell infiltration of the uveal tissue.[1]

The following study was undertaken to determine whether plasma cells are responsible for antibody production in the uveal tissue of the inflamed eye. Coons and coworkers [2] have shown that antibodies within cells can be made visible by a variation of their method, using fluorescein-labeled antibody. Frozen sections of tissues containing antibody are dipped in a dilute solution of homologous antigen, then carefully washed, and the precipitated antigen demonstrated by means of specific fluorescein-labeled antibody. With this technique they showed that following the subcutaneous injection of egg albumen, human gamma-globulin, or diphtheria toxoid, typical plasma cells of the regional lymph nodes and spleen contained high concentrations of antibody. The present investigation deals with the problem of antibody production in the uveal tissue after the intravitreous injection of crystallized egg albumen. The enhancing effect on antibody formation by caterpillar hair as a local irritant in the anterior chamber of the eye, previously described, was utilized in two experiments. Serum and aqueous humor were tested for the presence of antibodies, and the antibody activity was determined. Frozen sections of the eye were stained for antibody by pretreatment with diluted egg albumen and staining with fluorescein-labeled anti-egg-albumen rabbit serum.

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MATERIALS AND METHODS

Crystallized hen-egg-albumen (Armour & Co.) was used throughout these experiments. Injections of 0.15 ml. of a 4% solution in 0.9% NaCl were made into the vitreous body and of 0.15 ml. into the anterior chamber of one or both eyes of six normal albino rabbits, with an average weight of 4 to 6 lb. In those rabbits in which only one eye was injected the total amount of egg albumen was 10-15 mg.; if both eyes were injected, it was 20-30 mg. Caterpillar hair of Malacosoma americana served as a local irritant. A tuft of 10-20 sterile hairs was introduced into the anterior chamber of both eyes of two rabbits 15 days prior to the egg albumen injection, using a technique described in an earlier paper.[1] Two rabbits received a second injection of 0.2 ml. of a 2% egg albumen solution (i. e., 4 mg.) into the anterior chamber 15 days after the first injection. Antibodies in the serum and aqueous humor were determined by means of a hemagglutination technique with tannic-acid-treated red cells, according to Stavitsky.[3] The gamma-globulin contents of the serum and of the aqueous humor were determined by paper electrophoresis, and the antibody activity was calculated as the reciprocal of the agglutination titer per gram of

gamma-globulin. The eyes were removed at different time intervals (1 to 29 days) following the injection of antigen. Immediately after enucleation the eyes were bisected sagittally and the lens and vitreous body carefully removed. Half of the globe, including part of the cornea, iris, ciliary body, sclera, and choroid, was then placed in a test tube, quick-frozen in an alcohol-dry ice mixture and stored at —20 C. The other half was fixed in Bouin's fluid for histological study. The frozen eyes were cut in 6μ sections according to the technique of Coons and co-workers.* The freshly cut sections were dried at room temperature, fixed in 95% ethanol at 37 C for 15 minutes, dried again in an incubator for 30 minutes, and treated with a 1:1000 solution of egg albumen in buffered saline for 30 minutes. The sections were washed with saline, covered with a drop of antibody conjugate (fluorescein-labeled anti-egg-albumen serum of rabbit prepared according to the technique of Coons and Kaplan [5]), and placed in a moist chamber. After 30 minutes they were washed again with saline and mounted with a drop of buffered glycerol. Control sections were treated with saline instead of the antigen solution and stained later with the same antibody conjugate. Since antigen was still present in many of the eyes examined, tests for the specificity of the antibody staining were carried out by pretreatment of the sections with unlabeled anti-egg-albumen immune serum for 30 minutes prior to the staining with antibody conjugate in order to inhibit the specific staining of the antigen. The stained sections were examined with the fluorescence microscope, as described by Coons.[4] The number of antibody-containing cells in the uveal tissue was tabulated and expressed in symbols (+++ and ++, Table). For purposes of comparison and identification of the infiltrating cells, other sections were stained with the Giemsa method and examined microscopically.

* References 4 and 5.

RESULTS

After the injection of 15 mg. of egg albumen into the vitreous body and anterior chamber, all rabbits showed a very marked reaction, consisting of an acute fibrinous iritis. The signs of inflammation subsided slowly, and at the time of enucleation (1 to 29 days after the injection) all animals exhibited a distinct Tyndall effect in the anterior chamber and hyperemia of the iris.

The Table contains the data on hemagglutination and antibody activity and on the presence or absence of antibody and antigen in the sections. As in previous experiments with other antigens, there was marked individual variation in the experimental animals. On the fourth day after a single injection of 15 mg. of egg albumen into the vitreous and anterior chamber (Rabbit 5) there was no antibody detectable by hemagglutination, either in serum or in aqueous humor. Accordingly, no fluorescent cells were found in the tissue sections of the right eye, but antigen could be seen. On the seventh day low antibody activity was noticed in the serum and aqueous humor and a few fluorescent cells were found in the sections of the left eye. At a later date (14 days) in another rabbit (#6) the many fluorescent cells visi-

Antibody Activity of Serum and Aqueous Humor and Presence of Antibody-Containing Cells in the Uveal Tissue After Intraocular Injection of Egg Albumen

Rabbit No.	Days After Injection	Observed Titers		Gamma-Globulin Content*		Antibody Activity		Microscopic Presence of	
		Serum	Aqueous	Serum	Aqueous	Serum	Aqueous	Ab †	Ag ‡
5	4	...	...	....	....	...	...	—	+
	7	40	10	1.61	0.92	40	10	+	(+)
6	14	80	1,280	1.55	1.17	50	1,000	+++	+
2	15/14	40	160	1.68	1.20	24	130	+	(+)
	22/8	640	640	1.62	0.85	400	750	+++	(+)
4	20/6	1,280	640	0.96	0.61	1,330	1,050	++	(+)
	29/14	1,280	320	1.04	0.58	640	550	++	—
2	3	...	320	1.34	1.17	...	270	—	(+)
	7	640	640	1.14	0.97	500	600	++	+
1	13	160	160	1.45	1.79	110	90	+	—

* Gamma-globulin content is expressed in grams per 100 ml.
† Ab indicates antibody. +++ means many fluorescent cells; ++, moderate number of cells; +, few cells.
‡ Ag stands for antigen: + = presence; —, absence.
§ 15/1 indicates 15 days after the first, and 1 day after the second, injection.

ble in sections of the injected eye (Fig. 1) paralleled the very high antibody activity in the aqueous humor. The noninjected (control) eye had no agglutinins in the aqueous humor and no antibody-containing cells in the uveal tissue.

Repeated injections of egg albumen in Rabbits 3 and 4 produced higher antibody activity in the serum and aqueous humor. In Rabbit 3 there was definitely higher activity in the aqueous humor, while Rabbit 4 showed no significant difference between serum and aqueous humor. In all these four eyes many fluorescent antibody-containing cells were present in the iris and ciliary body (Fig. 2).

Rabbit eyes (Rabbits 1 and 2) treated with caterpillar hair prior to the egg albumen injection showed also higher antibody activity. In the right eye of Rabbit 2 antibodies were already detectable by hemagglutination in the aqueous humor on the third day after the injection, while no antibodies were found in the serum. Surprisingly, no fluorescent cells could be found in a careful search through 10 sections of this eye. In the left eye, however, four days later, many antibody-containing cells were visible, mainly in the granulomatous tissue around the caterpillar hair, but also scattered throughout the stroma of the iris (Fig. 3). Despite this, the antibody activity of the aqueous humor was not significantly higher than the activity of the serum in the latter samples.

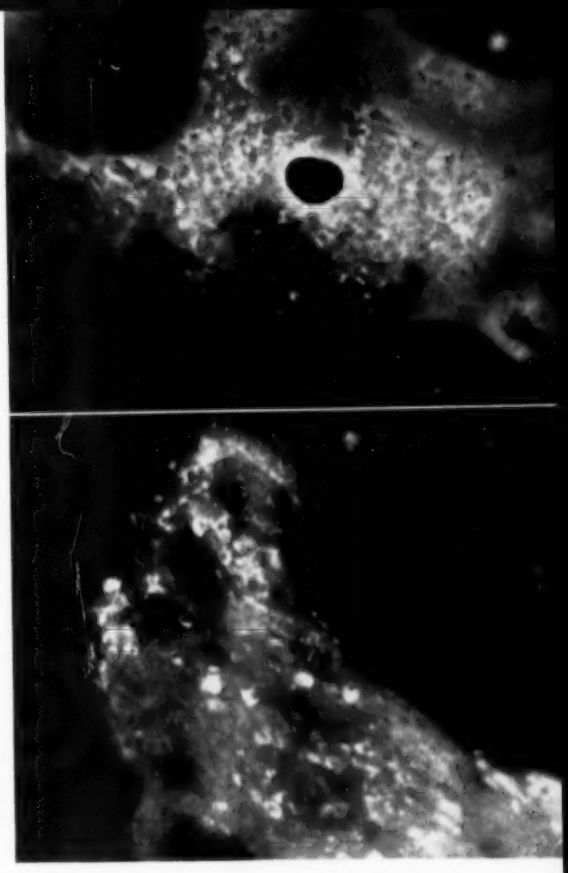
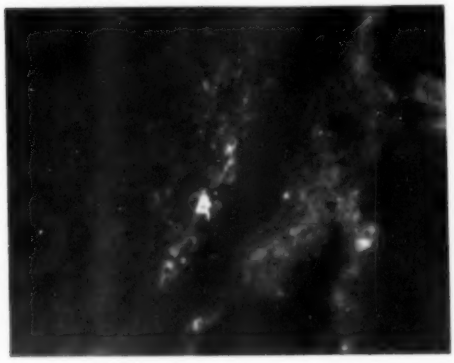


Fig. 2.—Upper (Rabbit 3): Reduced slightly from mag. × 200. Antibody activity of serum 400; of aqueous 750. Focus of infiltration by antibody-containing plasma cells in iris stroma around capillary.

Lower (Rabbit 4): Reduced slightly from mag. × 200. Antibody activity of serum 640; of aqueous 550. Ciliary process infiltrated by antibody-containing plasma cells.

Fig. 1 (Rabbit 6).—Reduced to 84% of mag. × 200. Antibody activity of serum 50; of aqueous 1000. Many fluorescent antibody-containing plasma cells in deep layers of iris.



In the control sections of eight eyes, small, fluorescent, mainly extracellular particles were found (Fig. 4). These sections had been treated with saline and antibody conjugate without dilute antigen solutions as a pretreatment. Some of these particles were artifacts, but there was also found diffuse staining of a few epithelial cells and mesenchymal cells of the iris stroma and ciliary body. The same particles and cells appeared in the sections stained for the presence of antibody by means of antigen and fluorescein-labeled antibody. It was thought that they represented antigen. All sections showing this phenomenon were therefore

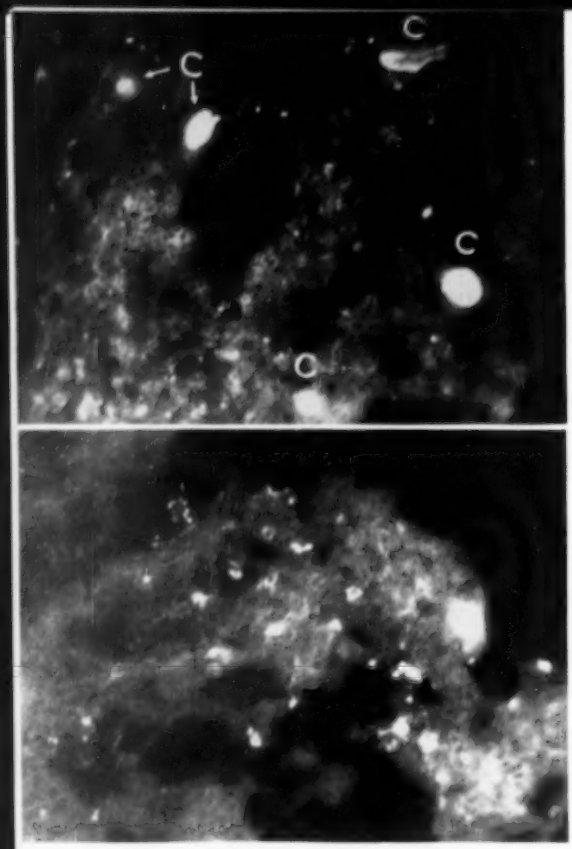
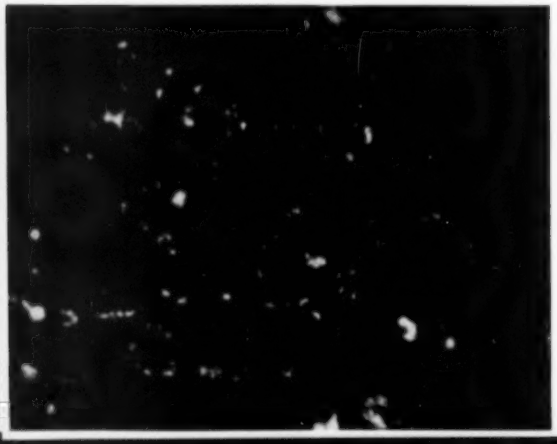


Fig. 3.—Upper (Rabbit 1): Reduced slightly from mag. × 200. Antibody activity of serum 110; of aqueous 90. Granuloma around caterpillar hair (C) containing many fluorescent plasma cells.

Lower (Rabbit 2): Reduced to 95% of mag. × 200. Antibody activity of serum 560; of aqueous 660. Scattered fluorescent plasma cells in iris stroma.

pretreated with unlabeled immune serum in order to neutralize at least part of the antigen. After 30 minutes these slides were stained with fluorescein-labeled antibody. In all instances a definite decrease of fluorescence of the extracellular particles and disappearance of the fine intracellular fluorescence were noted. This inhibition strongly suggests that this fluorescence was due to antigen.

Fig. 4.—(Rabbit 3): Reduced to ⅝ of mag. × 200. Antibody activity of serum 24; of aqueous 130. Extracellular fluorescent granules in vitreous; antigen.



Furthermore, it was noticed that the brilliantly fluorescent cells containing antibody were mainly found in the deeper layer of the iris, sometimes in small foci of infiltration, or in the granulomata of the two eyes treated with caterpillar hair. Examination under higher magnification in the fluorescence microscope gave the impression that these cells were plasma cells; this was confirmed by staining control sections with the Giemsa method (Fig. 5).

Some of the eyes with high antibody activity in the aqueous humor showed diffuse fluorescence, due to antibody in the deeper layers of the cornea, but there was no cellular fluorescence; the antibody seemed to be adsorbed to the fibers and to Descemet's membrane (Fig. 6).

COMMENT

Experimental data obtained in rabbits' eyes after injection of egg albumen confirm that antibody in the uveal tissue is present in the infiltrating plasma cells. The mesenchymal cells of iris and ciliary body were not found to contain antibody; neither were the epithelial cells nor the corneal cells. In some cases of high antibody activity of the aqueous humor antibody was adsorbed to Descemet's membrane and to the fibers of the corneal stroma. There seemed to exist a general correlation between the antibody activity of the aqueous humor and the number of antibody-containing cells in the uveal tissue. Eyes with high agglutination titers in the aqueous humor showed many fluorescent plasma cells in the iris and ciliary body. In only one eye, showing moderate antibody activity after pretreatment with caterpillar hair, could no fluorescent plasma cells be found.

On the other hand, there was evidence that after intraocular injection of egg albumen, antibody formation was not limited

to the inflamed uveal tissue alone, but that there probably was systemic formation as well, since considerable amounts of antibodies were present in the serum of the rabbits (e. g., Rabbit 3 and, particularly, Rabbit 4). Very similar results have been reported by Seegal and Seegal[7] in their studies on local organ hypersensitiveness. They found that after intraocular injection of typhoid vaccine antibodies appeared in the serum as early as the 4th day, but not until the 12th day in the aqueous humor. Later, after six weeks, tissue fluids of the eye showed higher agglutination titers than the serum. No protein determinations were carried out by these authors.

Remembering that antibody could be demonstrated only in the cytoplasm of plasma cells lying deep in the stroma of the iris or in small foci, one must consider the possibility that a great part of this antibody, liberated from the plasma cell, does not necessarily find its way into the aqueous humor of the eye, but may well be absorbed into the blood stream within the iris and ciliary body and, therefore, appear later in the serum. The antibody found in the aqueous humor may be due partly to penetration of plasma cells into the posterior and anterior chambers and partly to diffusion of gamma-globulins from the blood into the aqueous humor. Thus, the quantity of antibodies determined in the aqueous humor does not necessarily reflect the total antibody formed in this eye. On the other hand, it might be that in special cases the entire antibody formation takes place in the inflamed eye; in such instances the local inflammatory process would be the source of the antibody found in the serum, as has been pointed out by Goldmann and Witmer.[8]

The method of fluorescein-labeled antibody has therefore provided new aspects in the question of antigen and antibody presence within the eye, as was predicted by Ashton[9] in his report on allergy and uveitis. There can be little doubt that reactions between antigen and antibody actually take place in the eye. It will be the task of future research to determine the many possible factors involved in such reactions.

Fig. 5.—Reduced slightly from mag. × 400. Antibody containing fluorescent cells in iris stroma, showing plasma cell structure.

SUMMARY

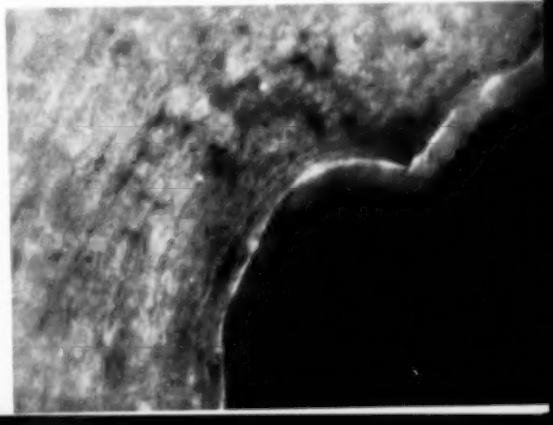
It was shown that local antibody formation takes place in the inflamed eye after injection of egg albumen into the vitreous and into the anterior chamber.

By use of fluorescein-labeled antibody combined with pretreatment of the frozen sections of uveal tissue with a dilute antigen solution, it was found that the cells containing antibody are plasma cells.

There was a rough correlation between the antibody activity of the gamma-globulins of the aqueous humor—determined by means of hemagglutination and paper electrophoresis—and the number of antibody-containing plasma cells in the uveal tissue.

Caterpillar hair in the anterior chamber as a local irritant seemed to act as a slightly enhancing factor in antibody production, and antibody-containing cells were found in the granulomata around the hair.

Fig. 6. (Rabbit 3).—Reduced to 5/6 of mag. × 200. Antibody activity of serum 400; of aqueous 750. Diffuse fluorescent staining of the cornea fibers and Descemet's membrane.



Dr. Albert H. Coons gave me the facilities of his department and instructed me in the immunological staining technique. Mrs. Jeanne M. Glancy-Connolly gave technical assistance.

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pHisoHex Keratitis

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pHisoHex (pHisoderm with hexachlorophene) is widely used as a preoperative cleansing agent for ocular surgery. A review of the literature revealed no reports pertinent to the effects, favorable or unfavorable, of this bactericide upon the human cornea.

Our recent experience with the use of pHisoHex as a preoperative cleansing agent for the face and lids indicates that this preparation is capable of inducing chemical injury to the corneal tissues.

A study of pHisoHex keratitis is presented in three sections:

I. Report of clinical cases of pHisoHex keratitis

II. Observations on pHisoHex induced keratitis in the rabbit and dog

III. Histopathology of pHisoHex keratitis in the rabbit and dog

I. REPORT OF CLINICAL CASES OF pHisoHex KERATITIS

Following are the clinical reports of two cases of deep keratitis which developed in adult patients following the chance introduction of small amounts of pHisoHex into the companion eye during the preoperative cleansing of the face and lids for evisceration and enucleation. As the postsurgical recoveries of both surgical eyes were uncomplicated, the observations will be confined to the pHisoHex-contaminated eyes.

CASE 1.—C. H. (City-County Hospital # 116377). Evisceration of the left eye was performed June 20, 1954, on a 58-year-old white woman as the terminal management of an intractable and fulminating Bacillus pyocyaneus corneal ring abscess.

A preoperative preparation was made of the patient's face and lids with pHisoHex. The surgical eye was then irrigated; the companion eye was not irrigated, as pHisoHex contamination of that eye was not considered to have occurred. A protective pad was placed over the nonsurgical eye to avoid bacterial contamination and corneal abrasion.

At completion of evisceration, with the patient under general anesthesia, the nonsurgical eye was noted to be injected and the cornea slightly hazy. No corneal abrasion was observed. The injected eye was irrigated by the surgeon; chlortetracycline (Aureomycin) was instilled and a binocular dressing applied.

On the first postoperative day, the right globe appeared markedly injected and showed 3+ chemosis. The lid conjunctiva was congested, and there was marked blepharospasm. A slight mucoid discharge was present in the lower cul-de-sac. The cornea was generally hazy, and further examination was deferred because of the marked discomfort induced by lid elevation. Material for a smear and culture was taken. Chlortetracycline and polymyxin ointments were prescribed to be administered at two-hour intervals.

On the second postoperative day, lacrimation and blepharospasm of the right eye were pronounced. A 4+ mixed bulbar injection was present. There was a small amount of mucofibrinous exudate in the lower cul-de-sac. The cornea was generally hazy, with a large, centrally situated epithelial denudation occupying approximately three-quarters of the corneal surface. The limbal margin of the cornea was epithelized and clear. There were folds in Descemet's membrane. No focal corneal infiltrative changes or ulcerations were present. Chlortetracycline ointment was instilled, and the eye was patched. A diagnosis of deep chemical keratitis, probably pHisoHex-induced, was presumed. Vision was markedly reduced.

On the third postoperative day, the smear and culture returns showed only a few diphtheroids. The patient was placed on homatropine drops and antibiotic medication discontinued. The conjunctival injection was less. The area of denuded epithelium was decreased in diameter, and a sizable area of central deep stromal edema was present.

Over the next five days, the global injection regressed and the cornea became epithelized. Deep

From the Division of Ophthalmology, Southwestern Medical School of University of Texas.

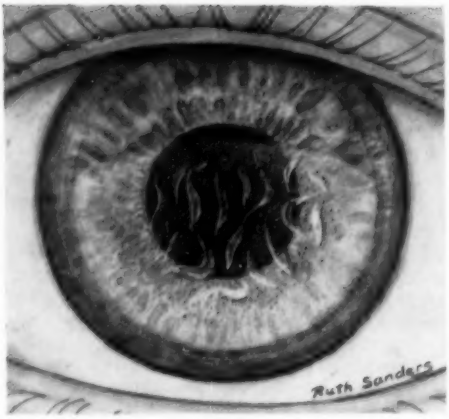


Fig. 1 (L. M.).—Nine days after pHisoHex. Diffuse illumination. Drawing.

stromal haze and edema persisted in the central two-thirds of the cornea.

Following are the reports of the biomicroscopic observations of the cornea made in the postoperative recovery period:

The first biomicroscopic examination, made on the second postoperative day, revealed a mottled haziness of Bowman's zone, composed of aggregates of punctate gray opacities. The central corneal stroma was edematous and thickened to approximately one and one-half times the clear periphery. The parenchymal swelling appeared to be localized in and slightly beyond the central area devoid of epithelium. The corneal stroma revealed interlacing and intersecting tracts and clefts of edema in all the lamellar planes, with increased intensity toward Descemet's zone. Descemet's membrane revealed many folds, corresponding to the area of stromal swelling and striation. The endothelium was clearly delineated peripherally, with bedewing on approaching the area of parenchymal swelling. The mosaic pattern became lost within the area of edema. The posterior central surface of the cornea was heavily sprinkled with fine, dust-like opacities. The anterior chamber revealed a 2+ flare, with many pigment particles in thermal circulation.

The second biomicroscopic examination, made on the fourth postoperative day, showed the central corneal haze area to be smaller, 7-8 mm. The central edema, mottling of Bowman's zone, parenchymal striation, and folds in Descemet's showed no change in intensity. The cornea was almost completely epithelized, but heavy superficial punctate stippling was present over the area of opacity. The central cornea showed endothelial swelling, most manifest axially, with an abrupt peripheral margination where normal mosaic resumed. Myriad punctate pigment aggregates could be seen on the posterior corneal surface within the edematous area. A few large solitary pigment spots were noted both within the opaque area and in the areas of clear endothelium.

On the sixth postoperative day, biomicroscopic examination showed only a small subcentral patch of unsurfaced cornea. The area of haze and striation with endothelial disturbance was still present in diameters extending considerably beyond the periphery of the residual epithelial denudation, with intraepithelial opacities and staining superficial punctate dots centrally.

On the seventh postoperative day, biomicroscopic examination showed no remaining areas of epithelial denudation on the corneal surface. The general corneal haze was much reduced, and the swelling of the central parallelopiped was gone. There was increased relucency of Bowman's membrane and of the posterior pre-Descemet stroma. Descemet's membrane appeared irregularly thickened with focal areas of relucency of variable size. The posterior surface of the cornea was dusted with pigment particles. There was an accentuation and irregularity of the endothelial mosaic, simulating the bronzing of the guttate cornea. Scattered superficial punctate epithelial opacities were present centrally, with a heavy superficial punctate epithelial staining. The patient still complained of sensitivity to bright light and intermittent global irritation.

Two weeks postoperatively, a superficial punctate keratitis, posterior stromal haze, and heavy Descemet's folds were still present. A few posterior corneal pigment spots were noted with some floaters and a faint flare. Vision was 20/20 in the affected eye.

Six weeks postoperatively, folds in Descemet's membrane still persisted. A guttate alteration of the endothelial mosaic was present, with a slight posterior stromal haze. There was scanty superficial

Fig. 2 (L. M.).—Nine days after pHisoHex. Focal illumination. Drawing.

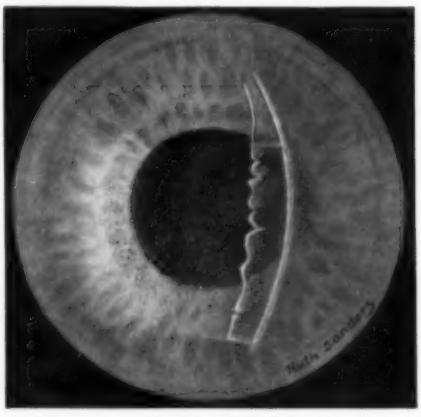
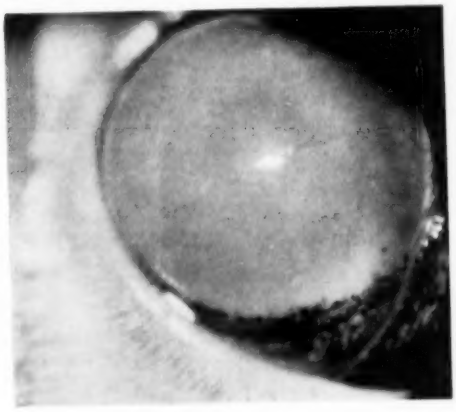


Fig. 3.—Photograph of rabbit eye. Three-minute pHisoHex exposure. Moderate opacity; extensive staining area of epithelial denudation.

punctate stippling, with an increase in the previously observed pigment dusting of the posterior corneal surface and anterior lens capsule. The patient still complained of intermittent global irritation, scratching, and light sensitiveness.

Eight months postoperatively the visual acuity was 20/15—1. The slit-lamp examination of the pHisoHex-contaminated eye showed the following features: a very faint stromal mottling, composed of fine gray aggregates in the superficial one-third of the stroma; an occasional small punctate pigment spot on the posterior corneal surface; a few scattered spots of endothelial retropouching, and a circularly distributed pigment ring on the anterior lens capsule, indicating the area of iris lens capsule contact during the iritic phase of the deep chemical irritation.

Fig. 4.—Photograph of rabbit eye. Five-minute pHisoHex exposure. Marked opacity; extensive epithelial denudation.



pHisoHex produced in Patient 1 a deep keratitis requiring several weeks to resolve, with symptoms of global irritation and photophobia still persistent at the six-week post-pHisoHex examination. At nine months after pHisoHex contamination the patient's symptoms were nil and the biomicroscopic residua were minimal.

Case 2.—L. M. (Methodist Hospital # 209376), a 45-year-old white nurse. Enucleation was performed on Nov. 10, 1954, at the patient's request, because of recurrent attacks of iridocyclitis and cosmetic disfiguration in an amblyopic eye with a long-standing retinal detachment which had been unamenable to repeated surgery.

Preoperative cleansing of the skin of the face and lids of the right eye was made with pHisoHex by the surgical nurse immediately before the operative

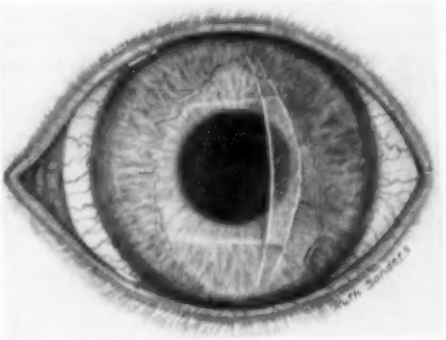


Fig. 5.—Drawing of rabbit eye. Three-minute exposure to pHisoHex. Moderate opacity; large area of epithelial denudation.

procedure, with irrigation of the surgical eye only. A protective eye dressing was placed over the lids of the companion eye to avoid corneal injury during surgery.

Enucleation of the right eye was performed, the patient being under general anesthesia, with installation of a buried implant. The surgery was uncomplicated.

Inspection of the companion eye at the end of surgery showed conjunctival hyperemia and slight corneal haziness but no corneal abrasion. The hyperemic eye was irrigated with saline and chlortetracycline ointment instilled. A monocular dressing was applied to the eye operated on.

On the first postoperative day, the examination of the left eye revealed a 3+ hyperemia of the bulbar and lid conjunctiva, with a 1+ global chemosis. The cornea was generally hazy, and the patient exhibited considerable photophobia and ble-

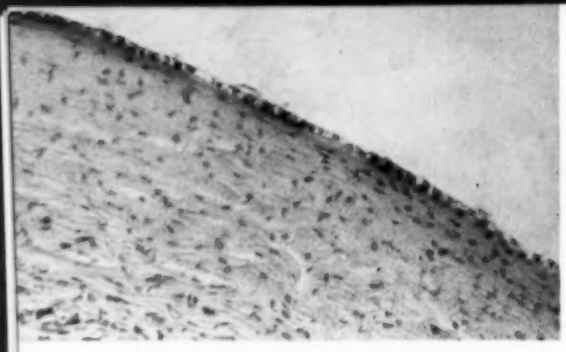


Fig. 6.—Photomicrograph from rabbit eye. Five-minute pHisoHex exposure. Hydropic endothelial changes.

pharospasm. No discharge was present in the cul-de-sac. No specific areas of corneal infiltrations were observed. The dressing was not changed on the enucleated eye and the patient was placed on treatment with chlortetracycline ophthalmic ointment three times a day in the affected eye.

On the second postoperative day, the left eye showed 3+ injection, with the corneal epithelium completely denuded save for a marginal epithelial cuff. The cornea had a 3+ general opacity, with heavy striation of Descemet's membrane. Cortisone ophthalmic ointment was instilled and a dressing applied.

In the subsequent postoperative days the global injection progressively resolved with treatment consisting of daily instillation of cortisone ophthalmic ointment and dressing occlusion of the affected eye.

By the fifth postoperative day the globe was pale and the cornea epithelized. A large area of corneal edema opacity, approximately 8 mm. in diameter, persisted centrally and subcentrally. Heavy striation of Descemet's membrane was still present, and a marked edematous thickening of the lower two-thirds of the cornea was present. The anterior one-third of the cornea was slightly hazy, with the predominant keratitic involvement occupying the posterior two-thirds of the stroma. The patient was able to count fingers through the peripheral clear portion of the cornea. A diagnosis of pHisoHex chemical keratitis was made at this time and 0.5% cortisone (Cortone) drops prescribed, to be instilled at two-hour intervals.

Fig. 7.—Photomicrograph from dog eye. Five-minute pHisoHex exposure; 24 hours later. Hydropic endothelial swelling.

On the eighth postoperative day, the patient was discharged from the hospital with a pale globe and persistent corneal edema opacification of the approximate diameters noted on the fifth day. The density of the opacity, however, had considerably decreased, and the patient was able to read medium-sized newsprint with effort.

On the ninth postoperative day, the first biomicroscopic examination was made. A central corneal edema approximately 6×8 mm. in diameter, with the long axis horizontal, occupied the subcentral area, with the upper extent of the haze overlapping three-fourths of the pupillary area. The area of opacity showed a marked fusiform edema swelling, resulting in a posterior focal thickening of the slit lamp beam in the opaque area of approximately one-third over the normal peripheral corneal thickness. The anterior one-third of the stroma was almost clear, with a generally smooth epithelial surface. A superficial punctate epithelial stippling was present centrally. The posterior two-thirds of the corneal stroma in the involved area showed considerable haziness with intensification of opacity at Descemet's level. The opacification of Descemet's membrane consisted of a diffuse, sheet-like haze, which was compounded by heavy opaque lines of Descemet's membrane folds. Myriad white dots were also localized at the level of Descemet's membrane. The endothelial mosaic could be distinguished in the clear peripheral areas but not within the area of opacity. A few cells were present in the aqueous. No keratic precipitations were noted within or at the margins of the corneal opacity. A drawing was made of the eye at this time by the medical artist. Vision at the time of drawing was 20/25 with best correction, not improved with a pinhole disc.

Fourteen days postoperatively the patient's vision was 20/20 in the left eye, with correction. The corneal edema was reduced 30% over the previous examination, and Descemet's folds were much less prominent. She still complained of photophobia in bright light.

On the 19th postoperative day, the corneal edema thickening was gone, with a faint visible haze persisting in the posterior cornea over the involved area. A few Descemet's folds remained.

Six weeks after surgery the corneal haze was reduced to biomicroscopic proportions, with no residual folding of Descemet's membrane in the area of involvement. A faint spotty mottling of the cornea was observable under high-power slit-lamp study and was present in both the anterior and the posterior portion of the stroma. A modified guttate alteration was present in the area of the previous opacity, with an irregular thickening of Descemet's membrane and spotty retroplacement of endothelial cell groups. A moderate pigment dusting was present on the posterior cornea centrally and inferiorly.

Biomicroscopic examination eight weeks postoperatively showed no change in the above observations. The patient still complained of light sensitivity in bright illumination and intermittent low-grade superficial irritation of the globe.

Three-month postoperative findings approximated those of the six-week observation, with increased clearing of the pre-Descemet stroma and marked decrease in foci of endothelial mosaic retroplacement. Symptoms other than bright light intolerance were nil.

Summary of Clinical Observations

pHisoHex can produce a severe keratitis involving all layers of the human cornea. A marked reduction of visual acuity occurs during the acute phase of corneal hydration. The clinical picture in the two cases presented was that of an acute chemical conjunctivitis, which readily resolved in a few days, associated with a persistent deep keratitis, requiring many weeks for complete regression.

The corneas in both cases observed showed pronounced haziness, densest centrally, with marked edema thickening of the involved portions. Only the most peripheral margins of the corneas remained transparent. The epithelium was almost completely denuded, with a limbal cuff remaining. Prompt epithelial restoration occurred within a few days, but a superficial punctate stippling persisted for several weeks. Heavy folds were present in Descemet's membrane, and in neither instance could endothelial mosaic be discerned centrally. Interstitial clouding and stromal edema thickening were most intense in the posterior two-thirds of the cornea and gradually receded over a five- to eight-week period. The edema regression appeared attendant upon the restoration of endothelial mosaic.

Although each patient recovered 20/20 vision in the affected eye, both complained of symptoms of photophobia and intermittent superficial irritation of the eye for months after the incident. In each case corneal biomicroscopic residua were present in the following form at four months after pHisoHex:

1. A faint focal mottling pattern of spotty stromal relucency, most evident in the anterior one-third of the stroma
2. A modified guttate alteration of the endothelial mosaic with irregularities of Descemet's membrane and focal retroplacement of endothelial cell groups
3. Faint pigmentation of the posterior central or more severely involved areas of the cornea

Nine months after pHisoHex the first patient showed only biomicroscopic sign 1 with signs 2 and 3 reduced to a degree compatible with physiologic variation.

II. Observations on pHisoHex-induced keratitis in the rabbit and dog

The objectives of animal experimentation with pHisoHex were threefold: (1) to evaluate the possible harmful effects of pHisoHex upon the corneas of the rabbit and dog; (2) to determine the threshold time for production of minimal corneal injury from pHisoHex contact in these animals, and (3) to identify the chemical component of pHisoHex which was injurious to the animal cornea.

Evaluation of pHisoHex Injury to the Rabbit and Dog Cornea

A. One drop of pHisoHex was instilled into the right eye of 10 rabbits. The eyes were not irrigated. All 10 rabbits showed the following 24-hour changes in the pHisoHex-exposed eye:

1. Conjunctival injection of moderate to severe degree with accompanying chemosis
2. Epithelial denudation averaging 40% of overall corneal surface loss
3. Epithelial and through-and-through stromal haze of a severity that grossly blurred the iris stromal pattern in the area of severest involvement
4. Hyperemia, moderate to pronounced, of the iris surface vessels

A biomicroscopically observable stromal haze residuum was still present at 14 days after pHisoHex exposure.

Fluorescein staining revealed superficial punctate keratitis still to be active 14 days after pHisoHex in 8 of 10 rabbits.

B. Two drops of pHisoHex were instilled into the right eye of 10 dogs. (Two drops were used because of the copious lacrimation

response of the dog, and the larger globe.) The eyes were not irrigated. At 24 hours the pHisoHex-exposed dog eyes showed variable degrees of conjunctival injection and keratitis, corresponding in type to that observed in the rabbit eyes. Six eyes showed focal epithelial denudation, while four eyes showed a marked superficial punctate-staining keratitis with patchy areas of confluence of punctate-staining groups. Some stromal haze was present in all eyes.

These eyes were not followed in daily observation, but four dogs were killed for histologic study of deep corneal changes produced by pHisoHex.

Determination of Threshold Time for Production of Minimal Corneal Injury from pHisoHex Contact

Rabbit Threshold Studies.—A. In this series 1 drop of pHisoHex was instilled into both eyes of each of 10 rabbits. At five minutes the eyes were copiously irrigated with 60 cc. of saline, taking care to wash out well the plica and lid cul-de-sacs. At 24 hours, 100% of the rabbit eyes showed keratitic changes similar to the nonirrigated pHisoHex-exposed eyes, varying only slightly in degree of severity of involvement. One rabbit in this series was killed at 24 hours for histopathologic study.

At 14 days after pHisoHex, all rabbit eyes but one still showed superficial punctate stippling and a biomicroscopically detectable stromal haze.

B. One drop of pHisoHex was instilled into each of 26 rabbit eyes and allowed to remain for one minute and the eye irrigated with 60 cc. of saline. At 24 hours, 18 eyes showed focal areas of corneal denudation with corresponding stromal haze, the areas varying from a small patch 2 mm. in diameter to one-half the corneal surface. Six eyes showed marked superficial punctate staining of the cornea, and two eyes showed no epithelial changes with clear corneas.

C. One drop of pHisoHex was instilled into five rabbit eyes with prompt irrigation of each eye at 30 seconds after pHisoHex instillation. These eyes showed no significant corneal changes other than an occasional slight stippling area with no corneal clouding.

Dog Threshold Studies.—A. Two drops of pHisoHex were instilled into the right eye of five dogs, and after five minutes of pHisoHex contact the eyes were irrigated with 60-70 cc. of saline. At 24 hours the pHisoHex-exposed eyes showed conjunctival redness and a keratitis similar to, but of less severe degree than, that in the dog eyes in the non-irrigated pHisoHex series.

Four of the five eyes showed a marked conjunctival hyperemia with focal corneal epithelial denudation and moderate corneal haze. The fifth eye showed a marked central superficial punctate keratitis but no gross areas of epithelial denudation.

B. Two drops of pHisoHex were instilled into 10 dog eyes with irrigation at four minutes. The 24-hour findings consisted of slight to moderate corneal edema opacity in nine eyes. Five corneas had focal epithelial denudation. Four eyes displayed heavy superficial punctate corneal staining, with patchy areas of confluence of many staining groups. One eye showed only minimal punctate staining.

C. Two drops of pHisoHex instilled into five dog eyes with irrigation at three minutes produced in all eyes only minimal superficial punctate staining with minimal to no corneal opacity.

Summary

Experimental studies with pHisoHex in rabbit eyes indicate that pHisoHex can rapidly, within one or two minutes, penetrate the rabbit cornea and produce a moderately severe keratitis, which endures for 10 to 14 days.

Little variation in severity of the keratitis was observed between the eyes irrigated after five minutes' pHisoHex contact and the nonirrigated pHisoHex-exposed eyes.

The threshold time for rabbit corneal injury, characterized by partial epithelial denudation and minimal corneal haze at 24 hours, appears to be approximately one minute, with 69% of eyes showing some epithelial denudation after 60 seconds' exposure.

A superficial punctate staining at 24 hours can be produced in the rabbit eye by as short a pHisoHex contact time as 30 seconds.

pHisoHex produces similar changes in the dog cornea. The threshold time for pHisoHex injury to the dog's eye was somewhat higher, possibly due to the copious tearing response of the dog's eye, and to a greater corneal resistance to pHisoHex penetration. The threshold for pHisoHex injury to the dog cornea appears to be approximately four minutes, with focal corneal denudation or patchy confluence of staining areas, and some degree of stromal haze developing in the majority of the eyes exposed for this period of time.

Identification of Chemical Component of pHisoHex Responsible for Injury to the Animal Cornea

pHisoHex (Winthrop-Stearns, Inc.) is essentially composed of (1) approximately 80% Entsufon,* a sulfonated ether (sodium octylphenoxyethoxyethyl ether sulfonate); (2) hexachlorophene, 3%, and (3) esters of hydrous wool fat U.S.P.

A. Ten rabbit eyes received 1 drop each of 3% pure hexachlorophene suspension in saline. The eyes were not irrigated. At 24 hours, nine eyes had clear corneas with no superficial punctate staining. One eye showed a small focus of minimal punctate staining.

B. One drop of trinitrotoluene (Triton, or pure Entsufon) was put in 14 rabbit eyes with no following irrigation. At 24 hours, all 14 eyes showed focal areas of corneal epithelial denudation with corresponding deep stromal haze.

C. Trinitrotoluene instilled (2 drops) into the right eye of 10 dogs produced 24-hour changes of focal epithelial denudation with corneal haze in 8 eyes. The remaining two dog eyes showed a superficial punctate keratitis, pronounced in one eye and mild in the other.

D. Hexachlorophene 3% (2 drops) placed in each of 10 dog eyes produced no corneal changes in 7 eyes and moderate superficial punctate stippling in 3 eyes.

III. Histopathologic Study of Animals

Six rabbit eyes showing characteristic 24-hour pHisoHex changes to a five-minute pHisoHex exposure were prepared in paraffin and stained with hematoxylin and eosin. The following general tissue changes were noted in multiple sections studied:

The corneal epithelium is missing in large areas. Some sections show stromal thickening in the area of epithelium and endothelium disturbance. The stroma is edematous, with interlamellar separation seen in many sections. Endothelial changes are present, varying in severity from a mild edema at the margins of the area of pHisoHex penetration to complete absence in some central portions. The endothelial alteration does not correspond entirely to the areas of epithelial damage, extending in some diameters beyond the point of epithelium loss peripherally. The endothelial cells are swollen by intracellular edema, with alteration of the nuclei to distorted forms of variable size and shape. A clear edema fluid lake surrounds the abnormal nuclei, distending the intact cytoplasmic membrane. In some areas the edematous endothelial cells are covered with a protein coagulum. The limbal transitional zones are variably infiltrated with inflammatory cells.

In the peripheral corneal areas, epithelium is intact, there is no stromal edema, and the endothelium shows a normal configuration with flat, oval nuclei. The ciliary body epithelium is normal, and there is a small amount of protein coagulum in the anterior chamber.

The iris vessels are engorged, but no cellular increase is evident in the iris stromal tissues. In the eyes where the lens capsules are preserved, there appears to be a slight intracellular edema of the subcapsular epithelium in a few sections. No consistent unequivocal subcapsular epithelial changes could be observed throughout the sections.

* Entsufon (trinitrotoluene; Triton) and pure G-11 hexachlorophene were provided by Dr. Thurber Guild, of Winthrop-Stearns, Inc., for this experiment.

Tissue studies of the pHisoHex-exposed eyes indicate that pHisoHex penetrates the rabbit and dog cornea, producing hydropic endothelial cell damage.

SUMMARY

Two clinical cases of pHisoHex keratitis are reported and described. The component of pHisoHex causing corneal injury is trinitrotoluene (Entsufon; Triton), a sulfonated ether. Trinitrotoluene can quickly penetrate the rabbit and dog cornea, producing a deep keratitis with epithelial loss and hydropic swelling and sloughing of the corneal endothelium.

The ultimate outcome of the corneal injury in the clinical cases observed was complete recovery of gross corneal transparency and visual acuity.

The threshold contact times for inducement of pHisoHex injury to the rabbit and dog cornea appear to be approximately one and four minutes, respectively. The threshold time for human corneal injury is not known.

COMMENT

The two cases of postoperative keratitis reported were resultant from pHisoHex contact with the corneas of nonirrigated companion eyes, throughout the period of surgery. The threshold of human corneal resistance to pHisoHex penetration is probably higher than that of the rabbit and dog. When one considers, however, the two clinical cases presented and the consistent production of keratitis in the rabbit and dog eye by a short preirrigation exposure of the cornea to pHisoHex, it becomes conceivable that at least an occasional postoperative deep striate keratitis may be pHisoHex-induced.

In intraocular surgery, the possibility of a residual dilute concentration of pHisoHex persisting in the conjunctival cul-de-sac following inadequate irrigation should be considered. Even minor concentrations of pHisoHex with direct access to the endothelium through the surgical incision would appear to be capable of producing endothelial damage.

The manifest treatment of pHisoHex keratitis is its prevention.

It is suggested that if pHisoHex is used as a preoperative bactericide on the skin about the eyes the following precautions be taken:

1. The surgical preparation should be made by the surgeon himself or by an assistant who has an intimate knowledge of the potentialities of the drug for producing severe ocular inflammation. Particular precaution should be taken to avoid ocular contamination in patients receiving general anesthesia.

2. No local anesthetic should be dropped into the eyes prior to the pHisoHex skin cleansing, thereby removing the patient's sensory protection against a pHisoHex burn. The eyes should be immediately irrigated if the patient complains of burning at any time during the cleansing procedure.

3. Both eyes should be routinely and copiously irrigated after pHisoHex lid and skin cleansing.

Mrs. Mildred Watkins and Mr. Robert Sage gave technical assistance in this work.

Contribution to the Histopathology of Cataract

JONAS S. FRIEDENWALD, M.D.
and
DOLORES RYTEL, Baltimore

Until quite recently a major puzzle in respect to the pathogenesis of radiation cataract has been the long lag period between exposure and the first visible signs of opacification in the lens. Von Sallmann * has shown that mitotic and other nuclear injury in the lens epithelium is an immediate effect of exposure to ionizing radiation, and Cogan[3] has found that these injured epithelial cells migrate posteriorly under the lens capsule, thus causing the posterior subcapsular disc-shaped opacity which is so characteristic of radiation cataract. There remains, however, a very large gap in our knowledge, for we have as yet no firm grounds for connecting these epithelial cell changes with the subsequent damage to lens fibers and the development of cortical opacities in the lens. The present study is an attempt to narrow this gap by seeking the earliest recognizable changes in the lens fibers.

The study of the histopathology of the lens cortex has been handicapped by the brittleness of this tissue on paraffin or celloidin embedding and by the relatively homogeneous appearance of the tissue with the usual histological stains. The interior of the lens fibers is essentially structureless except for the presence in some fibers of a cell nucleus. The chief cytological landmarks in the lens cortex must therefore be the cell surfaces of the lens fibers. Attempts to stain the cell surfaces with heavy metals, analogous to the silver stains of nerve fibers, have been made over at least seven or eight decades. Though occasional brilliant preparations have been achieved, the results of these stains have generally been patchy and irregular and complicated by large numbers of irregular and unpredictable artifacts. The substitution of copper for silver as the precipitated heavy metal [4] considerably improved the histological picture, the stain being limited to a lipoid-soluble (presumably phospholipid) component of the cell surface. However, the application of the copper technique to pathological problems has not, so far, been rewarding.

An opportunity for a new approach to this problem arose out of histochemical studies in our laboratory on alkaline phosphatase.[5] It was found that with fructose-1,6-diphosphate as substrate the reaction product, visualized as cobalt sulfide, was deposited on or near the cell surface of the cortical lens fibers. The nuclei of the lens bow and the nuclei and cytoplasm of the lens epithelium are also stained by this technique. It seemed feasible to use this technique for the study of the early morphology of cataractous changes. As compared with the heavy metal surface-staining procedures, this technique had the advantage of being employed on fresh frozen sections and, hence, of being free from fixation artifacts.

METHODS

Histological Technique

The histological technique used was that previously described. In adapting this technique to rat lenses, it was found necessary to increase the concentration of fructose-1,6-diphosphate to 0.16 M. The magnesium salt of this ester was substituted for the barium salt which was used in previous experiments, thus eliminating the conversion of barium to sodium salt.

Immediately after enucleation the eye was mounted in saline on the stage of a freezing microtome and 20μ sections were cut, using the technique

From the Wilmer Ophthalmological Institute of The Johns Hopkins University and Hospital.

This study was supported by a grant-in-aid from the Atomic Energy Commission.

* References 1 and 2.

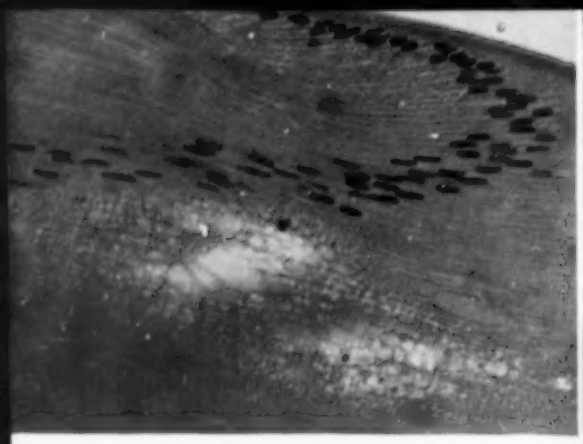


Fig. 1.—Celloidin section of rat lens with galactose cataract; courtesy of von Sallmann. Sagittal section through the lens bow stained with hematoxylin and eosin, showing hydrops of the lens fibers. Reduced to 90% of mag. × 250.

Incubation Mixture

Aminoacetic acid, KCl buffer, pH 9.4, in saturated sodium acetate solution	2.0 ml.
Saturated sodium acetate solution	2.0 ml.
$CaCl_2$ 4%	0.6 ml.
$MgCl_2$ 0.36 M	0.3 ml.
H_2O (glass-distilled)	0.1 ml.
Boiled kidney extract	1.5 ml.
Fructose-1,6-diphosphate (Mg salt)	1.5 ml.
Saturated Na_2FO_3	1 drop
Filtered after being incubated at 37 C for 1 hr.	

Fig. 2.—Fresh frozen section of rat lens with galactose cataract stained with the alkaline phosphatase method with fructose-1,6-diphosphate as substrate. Equatorial section showing hydropic lens fibers. Some of the swollen cells contain nuclei. Note that the boundaries of the hydropic regions show a double-layered membrane, indicating that these fluid accumulations are intracellular, not intercellular. Reduced to 90% of mag. × 500.

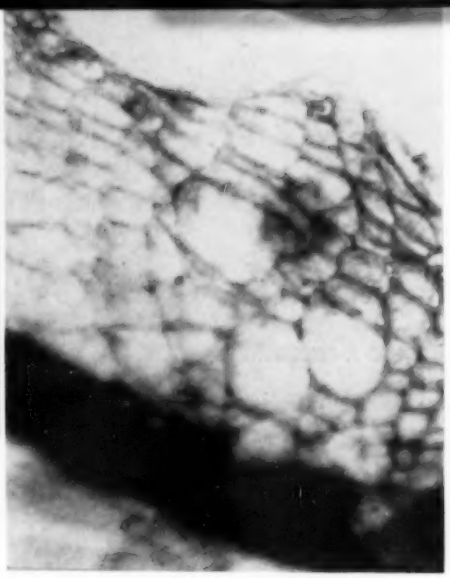


Fig. 3.—Same section as Figure 2. Reduced to 90% of mag. × 1000.

of White and Allen.[6] At the end of the desired incubation period sections were washed with a 1:1 solution of absolute alcohol and 4% $CaCl_2$. The procedures of Maengwyn-Davies and Friedenwald[7] were used for staining and mounting.

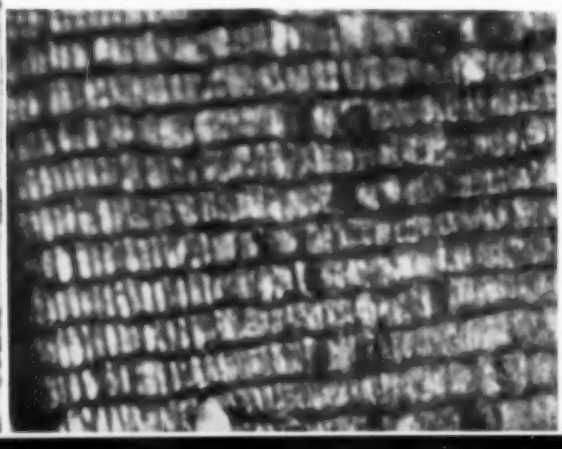
Galactose Cataracts

Female albino rats, weighing 150 gm., were placed on a diet of 50% galactose, 30% nonfat powdered milk, and 20% soluble starch. Bubbles or vacuoles in the subcapsular region near the lens equator were usually first seen 7 to 10 days after onset of the diet. The lesion develops rapidly, usually resulting in a complete cataract in approximately two weeks. Eyes were removed for study at three days and later, up to mature cataract formation.

Diabetic Cataract

After 48 hours of starvation, male albino rats, weighing 100 gm., were given intramuscular injec-

Fig. 4.—Early stage of galactose cataract, with only minimal hydropic changes in a few of the lens fibers. Section shows the equatorial layer of cells immediately under the lens capsule. Reduced to 90% of mag. × 500.



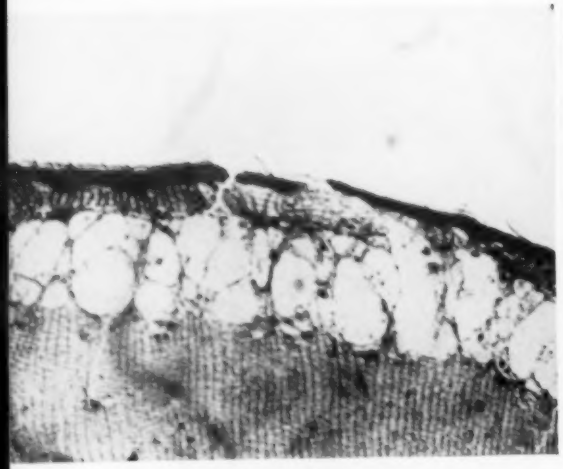


Figure 5

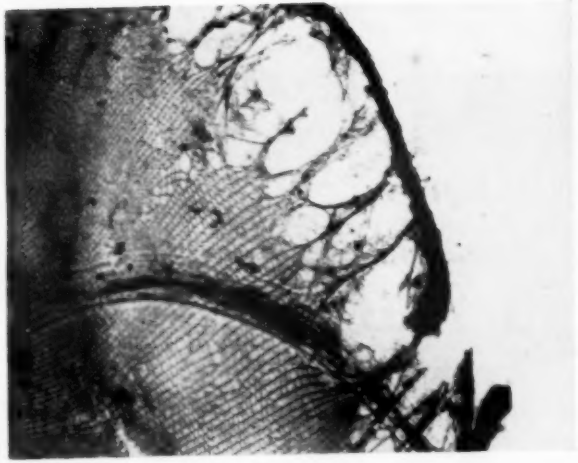


Figure 6

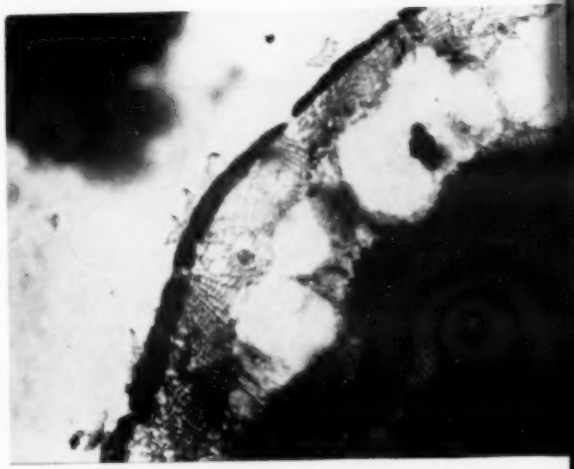


Figure 7

Fig. 5, 6, and 7.—Galactose cataract two weeks after onset of diet, with large vacuoles in the subcapsular cortex; sectioned and stained as in preceding Figures. Some of the vacuoles contain cell nuclei, indicating that the fluid is intracellular. Reduced to 90% of mag. × 100.

Figure 8

Fig. 8.—Galactose cataract two weeks after onset of diet. Same section as Figure 6, showing beginning hydrops of lens fibers deep in the cortex. Reduced to 90% of mag. × 250.

Fig. 9.—Alloxan diabetes cataract in a rat 10 weeks after onset of the diabetes. Fresh frozen equatorial section stained as in the preceding Figures, showing hydropic fibers in the subcapsular cortex. Reduced to 90% of mag. × 250.

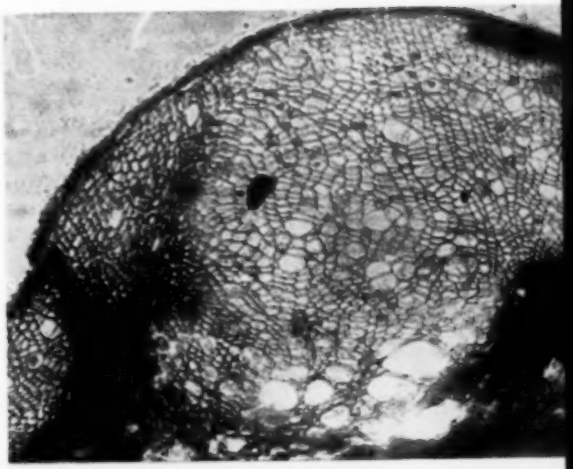


Figure 9

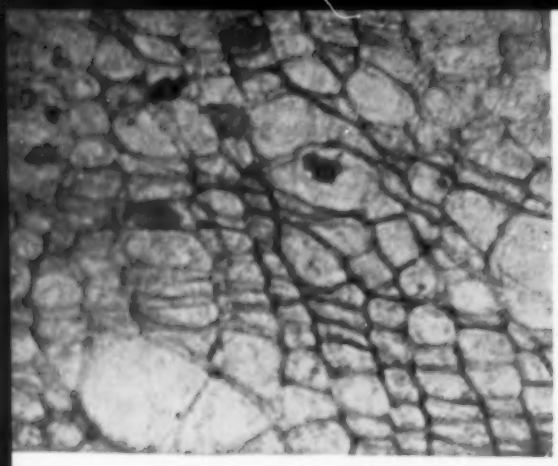


Figure 10

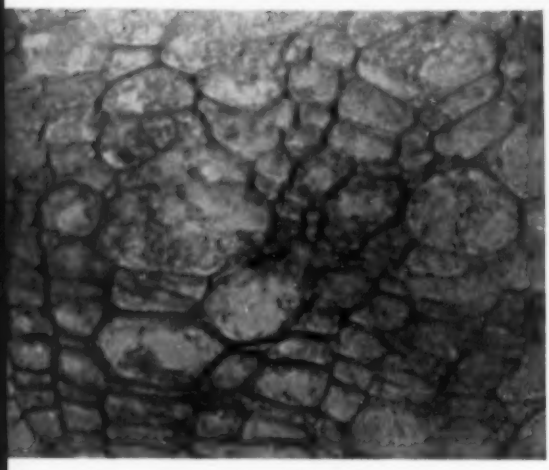


Figure 11

Figs. 10 and 11.—Alloxan diabetes cataract; same section as Figure 9, showing hydropic cells. Reduced to 90% of mag. × 1000.

tions of 35 mg. of alloxan. Two days after injection all animals excreted urine which was found to be 3+ to 4+ in sugar content. It was frequently necessary to reinject animals to sustain a diabetic condition during the incubation period of the cataract. After five weeks, 75% of the group showed equatorial and anterior subcapsular cataracts. Rats were killed at weekly intervals after this period for histopathological examination.

X-Ray Cataract

The right eyes of 100 gm. male rats were exposed to 2000 r of x-rays from an 85 kv., 5 ma. source screened through 5 mm. of aluminum, with an air gap of less than 2 in. (5 cm.). Total exposure of eight minutes was given in two four-minute runs, with a brief interval between to allow the tube to cool off. Exposure was through a hole in a 3 mm.

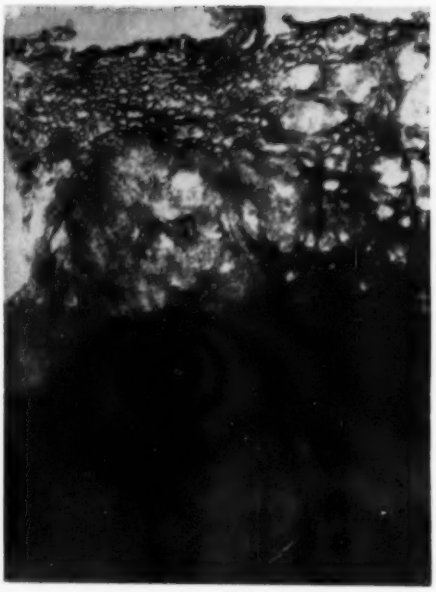
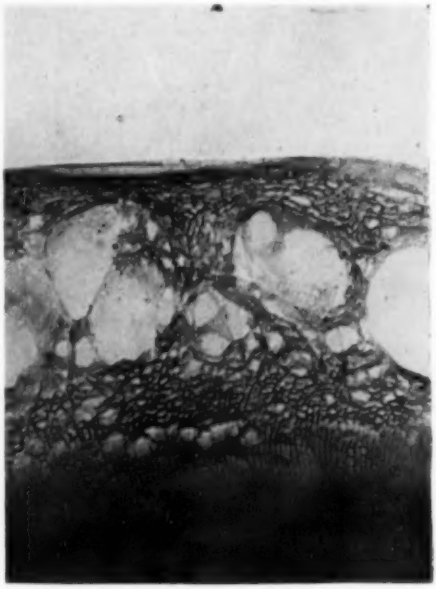


Figure 12

Figs. 12 and 13.—X-ray cataract in a rat five months after exposure to 2000 r. Fresh frozen equatorial section stained as in the preceding Figures, showing hydropic fibers in the subcapsular cortex. Reduced to 90% of mag. × 150.

Figure 13



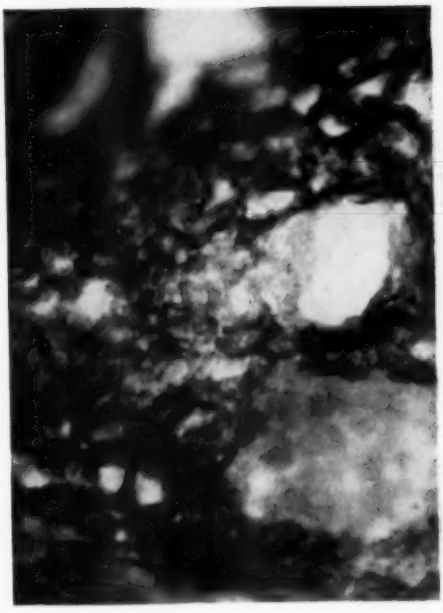


Figure 14

Figs. 14 and 15.—Higher magnification of the sections shown in Figures 12 and 13, showing confluence of some hydropic cells into a single pool. Reduced to 90% of mag. × 150.

Figure 15

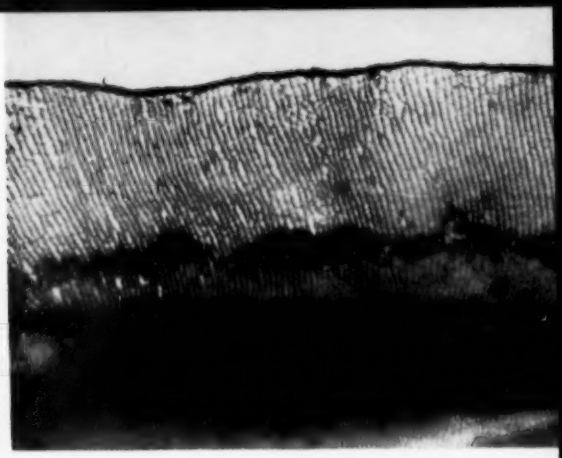


Fig. 16.—Normal control rat lens sectioned and stained as in the preceding Figures. Reduced to 90% of mag. × 100.

lead sheet, which protected the left eye and the rest of the animal's body. Depilation and dermatitis of the exposed eyelids resulted, reaching a maximum severity in three to four weeks after exposure and subsequently subsiding with relatively little gross damage. Keratoconjunctivitis, sometimes resulting in widespread corneal vascularization, was regularly present, reaching a maximum severity four to six weeks after exposure and generally clearing by the 10th to the 12th week sufficiently not to hamper observation of the lens. Fine, dot-like opacities were generally visible in the posterior subcapsular region by the time the cornea had cleared sufficiently to permit observation. Cortical opacities in the equatorial region were first noted 12-14 weeks after exposure and progressed steadily thereafter. The lesion was remarkably uniform in its appearance and progress in different animals. The left eyes of the animals showed no lesions.

RESULTS

The histological pattern revealed by the stain in normal rat lenses did not differ from that previously described in respect to rabbit lenses. In all three forms of experimental cataract hydropic lens fibers were to be found. These varied from cells just visibly larger than normal to some blown up to 100μ or 200μ in diameter. That these were really swollen cells, and not intercellular accumulations of fluid, could be demonstrated in two ways. In general, the histochemical reaction product appears to be deposited just beneath the cell surface, so that the common boundary between two cells is characterized by a double line. A similar double line is visible at the boundary of even the largest lesions seen. Moreover, in some of the swollen cells a cell nucleus was visible within the swollen area.

In the earliest stages of the cataracts the hydropic cells were found in a zone somewhat beneath the surface in the equatorial region of the lens. This zone was narrower and more deeply located in the galactose and diabetic cataract, and more diffuse and more superficial in the radiation cataracts. In animals in all three series killed at the time when ophthalmoscopically visible opacities and vacuoles were shortly to be expected, but when no opacities were as yet visible, some moderately swollen hydropic cells were found. No similar lesions were found in controls or in animals killed three to four days (galactose series) or two to three weeks (x-ray series) before visible opacities developed in other animals of the same group. It is concluded that hydrops of the lens fibers represents the earliest change so far recognized in the lens cortex in the development of cataract of the types studied.

Visualization of the hydropic change is not exclusively limited to the technical procedure used in these experiments. These same changes can, on occasion, also be seen in routinely embedded and stained specimens.

Fig. 17.—Normal control rat lens. Reduced to 90% of mag. × 500.

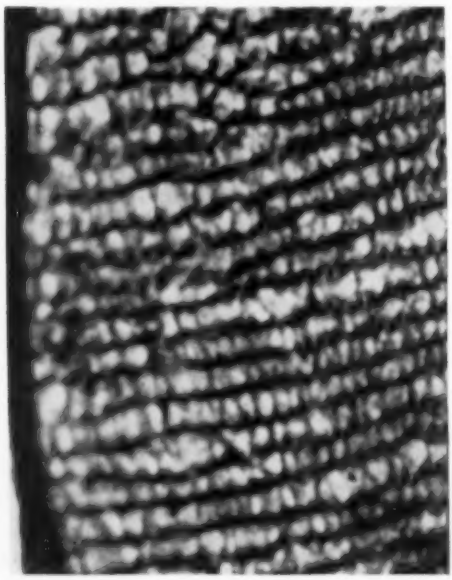


Fig. 18. Normal control rat lens, showing artifactual wavy thickness of section, due to knife vibration. Reduced to 90% of mag. × 100.

By permission of von Sallmann, we reproduce a hitherto unpublished photograph (Fig. 1), showing hydropic swelling of a lens fiber in the early stages of galactose cataract, embedded in celloidin and stained with hematoxylin and eosin.

Rupture and dissolution of the hydropic cells can be seen in the more advanced stages, leading to the interfibrillar clefts filled with protein coagulum, which are the common findings in cataractous lens cortex studied by routine techniques. The present study throws no light on the pathogenesis of this hydropic change.

SUMMARY

In these experiments an enzyme histochemical reaction has been used primarily as a morphologic technique to reveal early changes in the lens cortex in experimental cataract produced by galactose feeding, by diabetes, and by exposure to ionizing radiation. In all three types of experimental cataract, the earliest morphologic change noted in the lens cortex was hydrops of the lens fiber. The earliest appearance of the hydrops was

simultaneous with or only very shortly before the earliest appearance of ophthalmoscopically visible vacuoles and opacities in the lens cortex. It is interesting to note that the lens cortex was hydrops of the lens fibers. the phosphatase responsible for the histochemical reaction product is still active. Rupture and dissolution of the hydropic cells leads to the production of interfibrillar fluid clefts filled with protein debris.

No qualitative differences were noted in respect to the hydropic process in the three types of cataract studied, but the pattern of location of the hydropic cells in the early stages of the lesion was different in radiation cataract from that seen in the galactose and diabetic cataracts. This study throws no light on the pathogenesis of the hydropic change, but merely correlates hydrops of the lens fibers with the onset of visible opacities and vacuoles in the lens cortex in these types of experimental cataract.

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The Cells of Remak and the Astroglia of the Normal Human Retina

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The retina is a part of the central nervous system, and its neurons are supported by an elaborate system of glia, as are neurons in general. The supporting system of the brain contains three types of elements: the astroglia, the oligodendroglia, and the microglia; in the brain we possess a solid knowledge of all three types of cells. In the human retina, on the contrary, with the exception of the cells of Müller (radial fibers), not much is known of the glia. Friedenwald [1] stated that "unfortunately, few extensive studies have been made on the retina with special stains developed in the past 20 or 30 years to demonstrate neuroglia and microglia." This gap in our knowledge is due to technical difficulties, since with the conventional histologic procedures or the older metallic impregnations it is not possible to visualize the glia. However, some of these difficulties were recently alleviated by Scharenberg,[2] who perfected the silver carbonate technique of del Rio Hortega and developed a new astroglia variant which is very simple and reliable and permits the impregnation and photomicrographic recording of the astroglia of the C. N. S., including the retina under normal and pathological conditions.

In the present contribution we shall describe several types of glial elements of the normal human retina which have not previously been demonstrated.

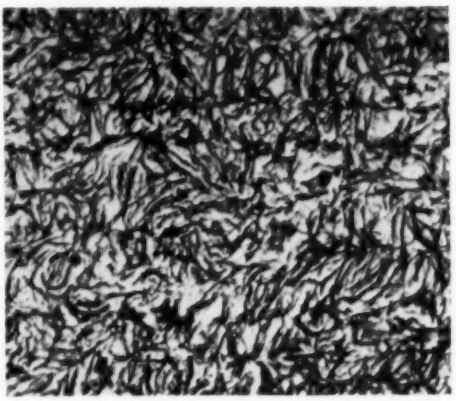

Fig. 1.—Horizontal section of the inner limiting membrane of the human retina with the mosiac-like pattern of the inner ends of the elements of Müller. Hortega method. Photomicrograph; reduced ⅔ of mag. × 700.

HISTOLOGIC DESCRIPTION

The order of our description follows the layers of the retina, proceeding from the inner to the outer layers.

The inner limiting membrane is considered as representing an acellular product of the retina (Kolmer [3]). This membrane is intimately connected with the elements of Müller, the brush-like inner ends of which find a firm anchorage in this membrane (Fig. 1) and penetrate the retina radially (Fig. 2). In horizontal sections of the retina these inner processes of the elements of Müller form a mosaic-like pattern (Fig. 1), suggesting that the limiting membrane and Müller's fibers form a skeleton which supports

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From the Laboratory of Neuropathology and Neuro-Ophthalmology, Neuropsychiatric Institute, University of Michigan.

Fig. 2.—Brush-like inner processes of the elements of Müller in a radial section of the human retina, forming the black-stained inner limiting membrane (*a*). (*b*) Nerve fiber layer; (*c*) ganglion cell layer; (*d*) inner plexiform layer; (*e*) inner nuclear layer. Hortega method. Photomicrograph; reduced to ⅔ of mag. × 600.

the nerve cells, various other glial elements, and the blood vessels. The inner surface of the inner limiting membrane is partly covered by a discontinuous sheet of scattered flat cells with long, interconnected processes, the nature of which is still obscure (Friedenwald).

In the layer of the optic nerve fibers, which are not myelinated, we were able to visualize numerous bipolar glial elements, with an elongated nucleus and two long, thin, and straight processes which accompany the nerves, but have apparently no connections with the brush-like inner processes of the elements of Müller (Fig. 3). These bipolar cells are identical with the elements of Remak, or lemmocytes (del Rio Hortega). They are not astrocytes, and their nature and significance will be discussed later. These elements were not found in the area of the optic disc, which contains numerous astrocytes in all of its layers (Marchesani,[4] Kolmer,[3] Polyak [5]).

In the ganglion cell layer there are very numerous well-differentiated astrocytes of a definite type: These are star-shaped elements with a round nucleus and several long and slender processes, which maintain an intimate and very characteristic relationship to the blood vessels and to the neurons (Fig. 4). These cells are arranged horizontally, surround the vessels with a dense network of

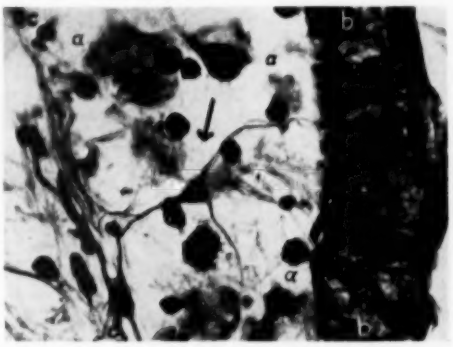


Fig. 4.—Single astrocyte (arrow) of the ganglion cell layer of the human retina sending its processes between ganglion cells (*a*) and large (*b-b*) and small (*c-c*) blood vessels. Horizontal section. Hortega method. Photomicrograph; reduced to ⅔ of mag. × 900.

Fig. 3.—Lemmocyte (fiber of Remak) of the nerve fiber layer of the retina (*a-a*). The nerve fibers are not impregnated. (*b*) Parts of the brush-like inner ends of the elements of Müller. Horizontal section. Hortega method. Photomicrograph; × 900.

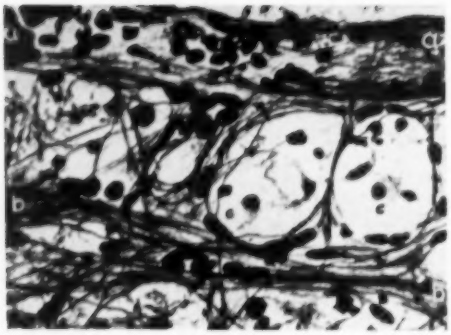


Fig. 5.—Dense network of processes of astrocytes in the ganglion cell layer of the human retina connecting blood vessels (*a-a* and *b-b*) and ganglion cells (*c*), which are only faintly impregnated. Horizontal section. Hortega method. Photomicrograph; reduced to ⅔ of mag. × 600.

Fig. 6.—Processes of astrocytes (*b*) of the ganglion cell layer of human retina forming arch-like structures parallel to the wall of a blood vessel (*a-a*) and surrounding this with large foot-like endings. The vessel wall is not stained, but the erythrocytes are visible. Horizontal section. Hortega method. Photomicrograph; reduced to ⅔ of mag. × 600.

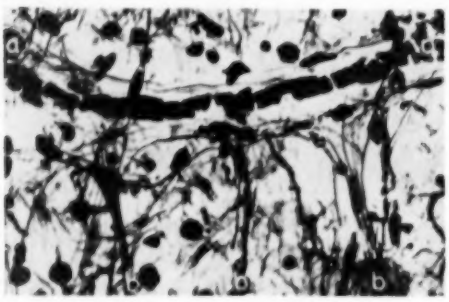
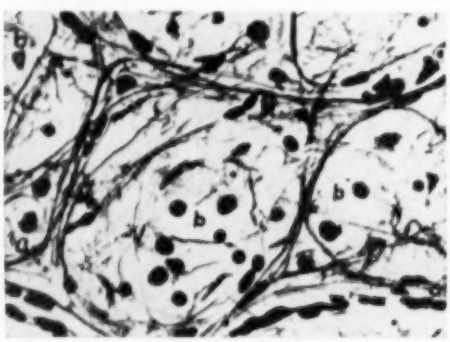


Fig. 7.—Honeycombed glial structures in the ganglion cell layer of the human retina. (*a*) blood vessels; (*b*) ganglion cells. Horizontal section. Hortega method. Photomicrograph; reduced to ⅔ of mag. × 600.



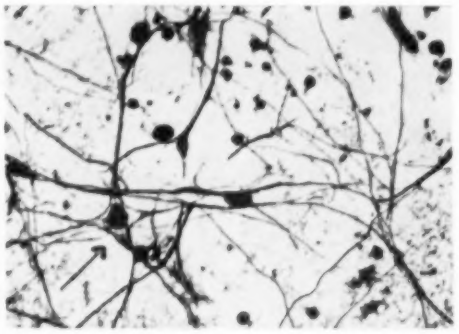
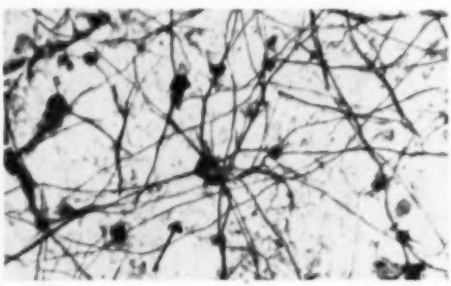


Fig. 8.—Astrocyte (arrow) of the inner plexiform layer of the human retina with its long and branched processes. Horizontal section. Hortega method. Photomicrograph; reduced to ⅔ of mag. × 600.

fibers (Fig. 5), and form arch-like structures parallel to the vessel walls (Fig. 6). Between the vessels of this layer numerous long processes of the astrocytes form an interconnected, honeycombed structure of remarkably regular pattern, which surrounds the nerve cells with a protective and supporting network (Figs. 5, 6, and 7). This network of the astroglia is placed at right angles to the coarser structures of the cells of Müller, which, as previously stated, are arranged radially (Fig. 2).

In the inner plexiform layer there are numerous astrocytes which are very similar in type to those of the ganglion cell layer: They are also star-shaped; their processes are numerous, long, and branched; they cross repeatedly and form a dense, horizontally arranged network, but the pattern of

Fig. 9.—Irregular network of the processes of the astrocytes in the inner plexiform layer of the human retina. Arrow indicates astrocyte. Horizontal section. Hortega method. Photomicrograph; reduced to ⅔ of mag. × 600.



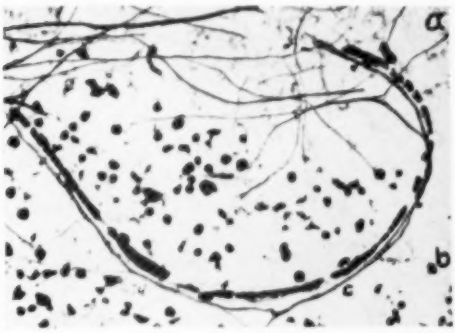
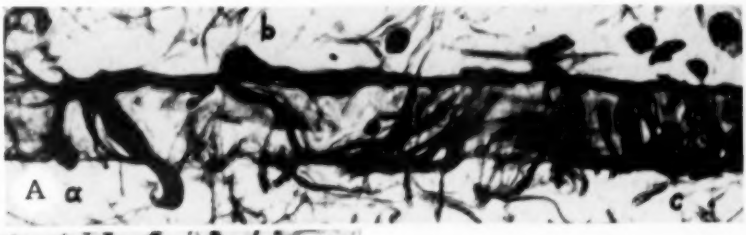


Fig. 10.—Processes of astrocytes of the inner plexiform layer (*c*) which enter the inner nuclear layer with a small blood vessel. The vessel wall is not stained, but the erythrocytes are impregnated. (*a*) Inner plexiform layer; (*b*) inner nuclear layer. Hortega method. Photomicrograph; reduced to ⅔ of mag. × 600.

this network is very irregular and differs greatly from that of the astrocytes of the ganglion cell layer (Figs. 8 and 9).

The inner nuclear layer contains no astrocytes of its own, but numerous processes of the astroglia of the inner plexiform layer enter it with the blood vessels (Fig. 10). The absence of astrocytes in this layer is not surprising, since most of the space be-

Fig. 11.—Dense interlacing network of fibers, which probably represent processes of astrocytes peculiar to the outer plexiform layer (*b*) of the human retina. (*a*) Inner nuclear layer, (*c*) outer nuclear layer. Hortega method. Photomicrograph; × 600.



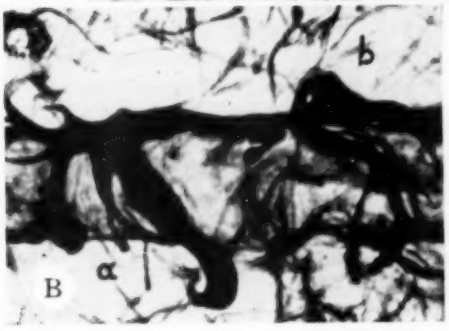


Fig. 12.—*A*, low power of a perivascular astrocyte on a blood vessel of the ganglion cell layer of the human retina. (*a*) Hand-like structure of the cell surrounding the vessel wall; (*b*) cell body, and (*c*) spiral-shaped processes of this astrocyte. Hortega method. Photomicrograph; × 500.

B, high power of the left part of the same astrocyte. × 900.

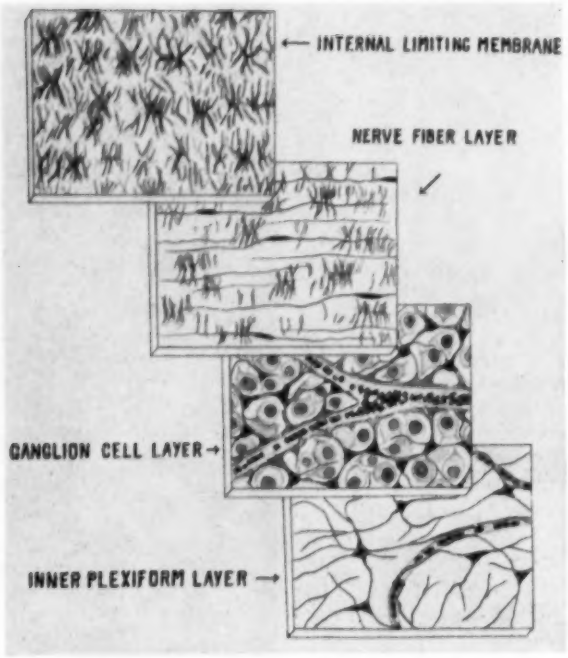


Fig. 13.—Scheme of the architecture of the glia of the four inner layers of the human retina. It does not show the astrocytes of the outer plexiform layer and the perivascular glia.

tween the neurons is occupied by the bodies of the cells of Müller, which are considered to be modified astrocytes (Marchesani [4]) or ependymal cells (Kolmer [3]).

In the outer plexiform layer the astroglia variant of the silver carbonate technique shows a dense, interlacing network of fibers, which probably represent processes of astroglia peculiar to this layer, but these fibers could not definitely be traced to the cell bodies (Fig. 11). There is no reason to identify these fibers with processes of ganglion cells or of the elements of Müller.

In the outer nuclear layer and in the layer of the rods and cones we could trace only the structures of the elements of Müller, which form the outer limiting membrane and the fibrillar baskets, but no astrocytes.

In addition to the perivascular types of astroglia above described, we found in the ganglion cell layer, and in close proximity to its vessels, an entirely new type of cell of a very complex structure: It has an elongated body placed on the vessel wall and numerous powerful processes which surround the vessels with irregularly spaced rings and spirals (Fig. 12 *A* and *B*). These elements seem to be present throughout the retina, and we have seen them also in other layers. They are similar to the spirocytes of del Rio Hortega * and Scharenberg.[5]

Figure 13 demonstrates schematically the architecture of the glia of the four inner layers of the retina but does not show the astrocytes of the outer plexiform layer and the perivascular glia, which require a further study because of their complicated structures.

COMMENT

Five new, morphologically different types of glia were found in the retina: (1) the lemmocytes of the nerve fiber layer, (2) the

* References 6 and 7.

astrocytes of the ganglion cell layer, (3) the astrocytes of the inner plexiform layer, (4) the astrocytes of the outer plexiform layer, and (5) the perivascular astroglia (spirocytes ?).

Detailed knowledge of the glia of the human retina has so far been restricted to the cells of Müller, including incomplete elements of this type (Ramón y Cajal,[9] Dogiel,[10] Marchesani,[4] Kolmer,[3] Polyak [5]). These authors used the techniques of Cajal, Ehrlich (methylene blue), the original method of del Rio Hortega, and that of Golgi; but these methods failed to impregnate the lemmocytes and the astrocytes, as described here.

As mentioned above, there were numerous lemmocytes (cells of Remak) in the nerve fiber layer. These elements were not demonstrated previously in the retina. According to del Rio Hortega [11] and Scharenberg,[12] these cells are intimately connected with the so-called "unmyelinated" nerve fibers of the peripheral nervous system and represent the equivalent of the elements of Schwann, which surround the "myelinated" nerves. The presence of the elements of Remak in the nerve fiber layer of the retina is not surprising, since its nerves are not myelinated.

Typical astroglia in the human retina has so far not been demonstrated. Kolmer [3] stated that there are no astrocytes in the retina proper, although they were found in the papilla of the optic nerve, and voiced the opinion that their physiologic tasks are carried out by the cells of Müller. Marchesani [4] expressed the opinion that the retina can be expected to possess an astroglia network but that the original astroglia method of del Rio Hortega failed to impregnate these cells. With the astroglia variant of Scharenberg [2] we were able to confirm this opinion of Marchesani and to visualize various types of astroglia in the retina without great difficulty. Polyak [5] mentioned radially arranged "fibrous and protoplasmatic astrocytes" in the retina of the chimpanzee and rhesus macaque (Fig. 85, Polyak [5]) but did not describe the dense, horizontally arranged network of astroglia which we found in three layers of the human retina.

The architecture of the astroglia in the retina is most closely adjusted to that of the neurons and the blood vessels and is of definitely different pattern in various layers. The elements of Müller form a coarser skeleton, between the structures of which the delicate network of the astroglia is spread, and both must, unquestionably, be regarded as vital supporting and protecting systems of the neurons.

The morphology of the perivascular astroglia and its relationship to the other elements of the blood vessel wall are not yet sufficiently studied. How intimate this relationship may be is indicated by Figure 12, which demonstrates an entirely new type of perivascular glia cell, with a large body and numerous processes coiled around the vessel. The significance of this type of glia cell and its place in the perivascular glial structures of the retina are still obscure.

SUMMARY

The histology of the normal human retina has been investigated by Ramón y Cajal, Dogiel, Marchesani, Kolmer, and Polyak. These authors described the morphology of the neurons and of the cells of Müller but failed to demonstrate the complete glial system.

With the improved silver carbonate technique of del Rio Hortega, it is possible to demonstrate various types of astroglia, which form an elaborate network, support the neurons, and surround the blood vessels. There are also lemmocytes (fibers of Remak) in the retina. The morphology of the glial system is adjusted to the requirements of the nervous tissue of the retina.

Photomicrographic evidence is submitted.

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Cyclodiathermy

A Clinical and Tonographic Evaluation

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This paper presents the results obtained in 38 eyes treated by cyclodiathermy using a technique described by Reiser * in 1949, and observed by us in Castroviejo's clinic in 1950. Tonographic studies were done postoperatively on 13 eyes in an attempt to gain some knowledge regarding the mechanism of action of this type of operation. An evaluation of cyclodiathermy seems appropriate at this time because the operation is experiencing a second peak of popularity, the first having occurred as a result of Vogt's † work in 1936. After his reports cyclodiathermy was widely used, but after a few years it fell into relative disrepute in this country.

Vogt's original method utilized as many as 100 punctures placed in parallel rows, beginning 2.5 mm. behind the limbus, over an area of the sclera extending one-third to one-half the circumference of the globe and reaching as far posteriorly as the insertions of the rectus muscles. The conjunctiva was first incised to expose the sclera to direct application of the electrodes. A 0.5 mm. needle was used in phakic eyes and a 1 mm. needle in aphakic eyes.

Vogt recommended the operation for glaucomatous eyes in which all other surgical and medical measures had failed, in glaucoma secondary to uveitis, and in hemorrhagic glaucoma. He found that keratitis or necrosis of the cornea with permanent corneal opacity could occur if the diathermy needle was placed less than 2.5 mm. from the limbus. Late iris hemorrhages also occurred as a complication. Vitreous occasionally escaped through the puncture holes at the site of application of the electrode, but he believed that this complication was not undesirable. Both favorable and unfavorable results from this technique have been reported in the literature. In this country, however, they were largely discouraging and the operation was gradually abandoned.

Our own results [7] with a small number of eyes (16) operated on from 1946 to 1948 were discouraging. The tension was controlled in seven eyes, but the occurrence of corneal edema and permanent corneal opacification in six eyes, probably due to endothelial damage at the periphery of the cornea, caused great concern. The angles of the anterior chamber, also, had been partially disorganized by the destructive process. We, therefore, discontinued use of the operation.

During the past five years, however, the development of new techniques for cyclodiathermy has stimulated renewed interest in the operation. Bietti [8] published an excellent review of the development of these methods in 1950. In nearly all of them the electrode is applied 6 mm. or more behind the limbus, a procedure which helps to eliminate the

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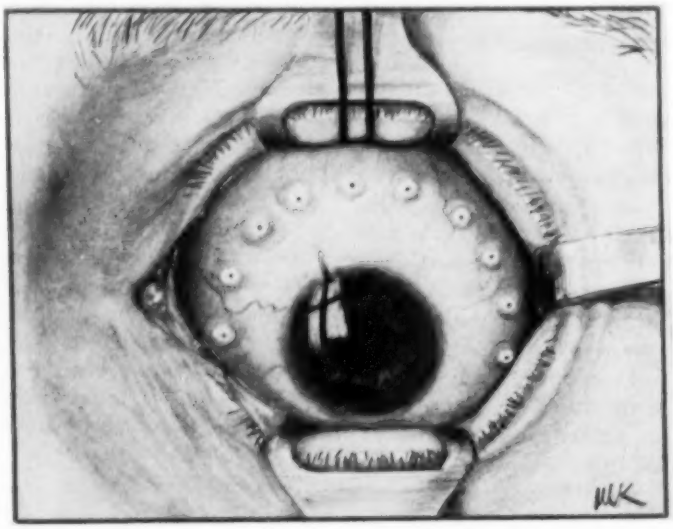
Read before the Section of Ophthalmology and Otolaryngology of the Houston Academy of Medicine, Jan. 28, 1955.

* References 1 and 2.

† References 3 through 6. References 5 and 6 are translated in the *British Journal of Ophthalmology* (**24**:288, 1940).

danger of damage to the periphery of the cornea, and the electrode is applied at only 10 or 20 sites. Each application is made for a period of 10 or 15 seconds, somewhat longer than in Vogt's method. Both surface and penetrating electrodes have been used. Albaugh and Dunphy [9] and L. Weekers and R. Weekers ‡ have been pioneers in the use of surface electrodes. The Weekers, seeking a method by which they might more accurately control the tissue temperature, developed a pyrometric surface electrode, which they applied 12 to 20 times or more to the conjunctiva 7 mm. from the limbus. Reiser,§ Arruga,[13] and others, in Europe, and Castroviejo,‖ in this country, have used perforating electrodes, which they applied through the intact conjunctiva 6 mm. or more back of the limbus. Guerry [14] coagulated the posterior ciliary arteries by applying the electrode along the visible course. Kettesy,[15] using a similar technique, coagulated both long posterior ciliary arteries just behind the insertions of the retracted horizontal rectus muscles. Arato,[16] in order to obtain better exposure, divided the muscles beforehand. To reduce the effects of diathermy on surrounding tissues, Diaz-Domingues [17] and Thiel [18] inserted a cyclodialysis spatula through the sclera into the suprachoroidal space and used it as an electrode to apply the electrical current directly to the ciliary body. These and other techniques not mentioned here provided a variety from which to choose.



Drawing illustrating the technique of cyclodiathermy used in this study.

The observations reported in this paper were all made upon eyes operated on after the method described by Reiser,¶ and employed in this country, at least for a time, by Castroviejo.#

‡ References 10 to 12.

§ References 1 and 2.

‖ Castroviejo, R.: Lecture given at the University of Florida Mid-Winter Postgraduate Course in Ophthalmology, Miami Beach, Fla., Jan. 18-20, 1951.

¶ References 1 and 2.

Castroviejo, R.: Lecture given at the University of Florida Mid-Winter Postgraduate Course in Ophthalmology, Miami Beach, Fla., Jan. 18-20, 1951.

TECHNIQUE

Retrobulbar and topical anesthesia are combined with the O'Brien type of akinesia. A 1.0 to 1.5 mm. electrode is applied through the conjunctiva 6 mm. behind the limbus at 8 to 10 points evenly spaced to cover slightly more than half the globe. The electrode is held in place for 10 seconds at each application. When diathermy of the upper half of the globe is desired, the punctures extend from the lower border of the internal rectus muscle to the lower border of the external rectus muscle (Figure). In this way, an attempt is made to obliterate the anterior ciliary vessels anterior to three rectus muscles and further to reduce the blood supply to the ciliary body by coagulating the long posterior ciliary vessels. As Vogt originally observed, occasional loss of a small amount of fluid from some of the punctures is harmless, and may even help to prevent secondary rises in pressure which might otherwise result from scleral shrinkage. In our experience, however, even if the tension is markedly elevated at the completion of the operation, it quickly compensates without complication. Only the eye operated on is covered, and the patient is not confined to bed. There is little postoperative pain, and the patient is usually discharged from the hospital in one or two days.

RESULTS

The method of cyclodiathermy just described was used for 38 eyes of 38 patients with glaucoma due to a variety of causes. Thirty-two of the eyes have been followed for a period of one to four years, and the remaining six, for a period of six months to one year (Table 1). In the treatment of primary glaucoma, the operation was usually used only when one or more previous operative procedures had failed or when ordinary filtration procedures were undesirable because of residual fields or because of the advanced age of the patient. It was also employed for eyes with infantile glaucoma and aphakic glaucoma after other operative procedures had failed, and for eyes with glaucoma secondary to aniridia and uveitis, conditions in which conventional operations ordinarily give poor results, or may even be contraindicated.

In this group of 38 eyes (Table 2), the ocular tension was controlled in 15 by cyclodiathermy alone, while the tension in 6 more was maintained at a normal level with the supplementary use of miotics. The tension could not be controlled in 17 eyes, either by repeated operation or by miotics. The operation was repeated one or more times in nine eyes and as many as four times in one eye without apparent untoward effect upon the globe. In reviewing our data, we find that the ocular tension in nearly all instances was reduced to normal for the first one to six months following operation, after which it again showed a tendency to rise. We feel, therefore, that the effectiveness of cyclodiathermy cannot be evaluated until at least six months, or even a full year, has elapsed since operation. Cyclodiathermy failed to control the ocular tension in any of the six eyes with infantile glaucoma, although in one of the eyes good control was maintained with supplementary miotics.

Although the operation has not been entirely successful in the control of ocular tension, it has, nevertheless, been a safe procedure in this small series of eyes. The one eye in which phthisis bulbi developed had previously been subjected to keratoplasty for scarring due to previous severe interstitial keratitis. Following the keratoplasty, severe secondary glaucoma necessitated an iridenclesis with sclerectomy. Another eye developed a rather severe postoperative iridocyclitis, which subsequently subsided. In the majority of patients, however, the operation caused surprisingly little reaction. No corneal opacities, intraocular hemorrhages, infections, or other complications were observed. We believe, in view of its safety and the fact that it controlled the ocular tension in 15 of 38 eyes without miotics and in 6 more with miotics, that it is a worth-while procedure for the ophthalmic surgeon to have available.

TONOGRAPHIC STUDIES

Several explanations of the mode of action of cyclodiathermy can be found in the literature. The operation may act by damage to the nerve supply of the ciliary body, causing alteration of axon reflexes; by diminution of the blood supply to the ciliary body, resulting in atrophy, or by changes in permea-

TABLE 1.—*Tabulation of Results of Cyclodiathermy in Thirty-Eight Eyes with Glaucoma*

Case No.; Patient	Age, Yr.	Diagnosis	Previous Surgery	Preop. Tension, Mm. Hg	Date of Operation	Type of Operation: No. of Punctures	Type of Operation: Site	Follow-Up	Result	Tension Controlled: With Miotics	Tension Controlled: Without Miotics
1 E. L.	47	Chronic simple glaucoma OS	Elliot trephine 11/7/45 Cyclodialysis 4/3/47	35	1/24/51	10	6 mm.	4 yr.	+	..	X
2 H. C.	57	Chronic simple glaucoma OD	Iridectomy	46	2/ 7/51	9	6 mm.	4 yr.	+	X	..
3 E. K.	60	Chronic simple glaucoma OS	Iridencleisis 10/18/50	40	3/21/51 6/20/51	9 9	6 mm.	3½ yr.	+	X	..
4 C. D.	67	Chronic simple glaucoma OD	None	46	3/21/51	9	6 mm.	4 yr.	+	..	X
5 B. P.	36	Aphakic glaucoma OD	Cyclodialysis 9/7/50	65	3/ 7/51	11	6 mm.	2 yr.	+	X	..
6 M. H.	53	Chronic simple glaucoma OS	Iridencleisis 5/1/50	48	3/ 7/51	11	6 mm.	4 yr.	0	..	..
7 D. B.	29	Glaucoma sec. to uveitis OS	Cataract extraction 3/14/51	60 28	4/25/51 9/ 6/51	9 9	6 mm. 6 mm.	3 yr.	0	..	..
8 H. S.	48	Aphakic glaucoma OS	Cataract extraction 1/5/43 Cyclodialysis 11/26/47	36 45	6/ 6/51 10/24/51	9 9	6 mm. 6 mm.	3 yr.	+	..	X
9 A. N.	70	Chronic simple glaucoma OS	None	62 40	6/12/51 3/ 5/52	10 10	6 mm. 6 mm.	3 yr.	+	X	..
10 A. F.	21	Glaucoma sec. to aniridia OS	Cataract extraction 7/12/51	34 34	9/ 5/51 10/10/51	10 10	6 mm. 6 mm.	3 yr.	+	X	..
11 J. W.	64	Chronic simple glaucoma OD	None	36 50 35	9/12/51 2/13/52 2/20/53	14 10 12	6 mm. 6 mm. 6 mm.	2 yr.	+	X	..
12 M. S.	51	Glaucoma sec. to alkali burn OS	None	60	9/19/51	10	6 mm.	1½ yr.	+	X	..
13 A. W.	54	Chronic simple glaucoma OD	None	30	10/10/51	10	6 mm.	3 yr.	+	X	..
14 J. H.	34	Glaucoma sec. to uveitis OD	None	60 60	10/24/51 2/27/52	10 10	6 mm.	3 yr.	0	..	..
15 L. D.	48	Aphakic glaucoma OD	Cataract extraction 4/19/51 Coagulation, retinal detachment 8/27/51	55	10/24/51	9	6 mm.	3 yr.	0	..	..
16 F. M.	68	Chronic simple glaucoma OD	None	35 62	10/24/51 12/ 5/51	10 14	6 mm. 6 mm.	1 yr.	+	X	..
17 R. M.	65	Aphakic glaucoma OS	None	40	10/31/51	10	6 mm.	3 yr.	+	X	..
18 L. B.	18 mo.	Congenital glaucoma OS	Goniotomy 11/14/50 Goniotomy and goniopuncture 7/21/51	56	12/ 7/51	12	6 mm.	2½ yr	0	..	..
19 L. W.	2½	Congenital glaucoma OS	Goniotomy Goniopuncture 11/22/49 Goniotomy and goniopuncture 2/2/50 and 6/15/50 Goniopuncture 9/22/50 Goniopuncture 12/15/50	47 47 40 Hard	2/ 2/51 5/11/51 12/ 7/51 4/11/52	13 9 10 14	6 mm. 6 mm. 6 mm. 6 mm.	3 yr.	0	..	..

20 A. D.	2½	Congenital glaucoma OD	Goniotomy and goniopuncture 3/25/51 and 6/12/51 Goniopuncture 9/7/51	52 55	11/30/51 2/ 1/51	12 10	6 mm. 6 mm.	3 yr.	0	..	..
21 S. T.	58	Chronic simple glaucoma OD	Corneoscleral trephine 11/28/50 and 10/2/51	40	12/11/51	12	6 mm.	15 mo.	+	x	..
22 N. H.	65	Chronic simple glaucoma OS	None	52	12/19/51	12	6 mm.	15 mo.	0	..	..
23 L. S.	62	Narrow-angle glaucoma OD	Iridectomy 3/30/50 Iridencleisis 6/21/50	60	1/ 2/52	8	6 mm.	14 mo.	0	..	..
24 F. B.	55	Chronic simple glaucoma OS	Goniopuncture 7/23/51 Corneoscleral treph. 8/29/51	55	2/20/52	13	6 mm.	3 yr.	0	..	..
25 L. R.	55	Glaucoma sec. to uveitis OD	None	55	2/20/52	10	6 mm.	3 yr.	0	..	..
26 S. C.	4	Congenital glaucoma OS	Iridencleisis Goniotomy Sphincterotomy Goniopuncture 8/23/51	40	2/14/52	12	6 mm.	13 mo.	+	x	..
27 W. C.	3	Congenital glaucoma OS	Combined 4/28/50 Combined 8/18/50 Goniopuncture 11/10/50 Combined 2/13/51	50	7/13/51	15	6 mm.	3½ yr.	0	..	..
28 T. K.	6	Congenital glaucoma	Goniopuncture 2/2/51 Combined 4/20/51 Combined 5/15/51 Combined 6/12/51	45	7/21/51	15	6 mm.	3½ yr.	0	..	..
29 N. H.	73	Glaucoma capsulare OD	Iridencleisis	40	4/30/52	11	6 mm.	2 yr.	0	..	..
30 J. A.	59	Chronic simple glaucoma OD	Cataract extraction 9/7/51 Retinal detachment 10/19/51	46	9/16/53	11	6 mm.	1 yr.	+	..	x
31 M. L.	67	Glaucoma sec. to uveitis OD	Cataract extraction 1932	80	10/ 1/53	7	6 mm.	1 yr.	+	..	x
32 B. B.	30	Glaucoma sec. to uveitis OD	Trephine	60	11/23/53	10	6 mm.	1 yr.	+	x	..
33 M. L.	69	Aphakic glaucoma OS	Cyclodialysis with peripheral iridectomy 3/11/54 Cyclodialysis 10/26/54	22	4/30/54	10	6 mm.	6 mo.	0	..	..
34 T. B.	54	Aphakic glaucoma OS	Intracapsular cataract extraction 10/8/52 Cyclodialysis 4/1/54	60	5/ 6/54	10	6 mm.	5 mo.	+	x	..
35 R. V.	69	Chronic simple glaucoma OD	Extracapsular cataract extraction 3/53	52	5/12/54	10	6 mm.	5 mo.	+	x	..
36 M. J.	56	Glaucoma sec. to uveitis OS	Basal iridectomy OD 6/49 Iridencleisis OS 8/50	57	5/12/54 OS	10	6 mm.	5 mo.	0	..	..
37 M. D.	73	Glaucoma sec. to herpes zoster ophthalmicus OS	None	62	5/12/54	12	6 mm.	5 mo.	+	x	..
38 C. B.	54	Glaucoma sec. to interstitial keratitis	Corneal transplant 12/4/53 Iridencleisis 4/29/54	38	9/16/54	10	6 mm.	2 mo.	Phthisis bulbi	..	..

TABLE 2.—*Summary of Results of Cyclodiathermy in Thirty-Eight Eyes*

(8-15 punctures; 6 mm. from limbus)		
Tension controlled		21
With miotics	6	
Without miotics	15	
Failure		17
Total		38
Complications		
Phthisis bulbi	1	
Iridocyclitis	1	
Corneal opacity	0	

bility either of the ciliary blood vessels or of the blood-aqueous barrier. Weekers and Prijot [19] have recently reported the results of tonographic studies indicating diminished aqueous production.

We have studied 13 of the eyes reported in this paper by tonography, but in only 2 eyes could these studies be made both before and after operation (Table 3).

Tonographic tracings done after all reaction to the operation had subsided demonstrated a considerably diminished production from that of normal eyes. The facility of outflow was also very poor, indicating that cyclodiathermy had decreased the production of aqueous. This was particularly well shown in one of the two eyes studied both preoperatively and postoperatively. We could not be certain but that these findings were compatible with obliteration of blood supply and atrophy of the ciliary body. In five of the eyes in which the ocular tension was not controlled by cyclodiathermy, the values obtained for the production of aqueous were all within normal limits. Of the two eyes subjected to tonography both before and after cyclodiathermy, the tension in one had been controlled without supplementary miotics, while in the other the operation had been a failure. In both these eyes the outflow values were diminished before and after operation, while aqueous production appeared markedly diminished postoperatively only in the eye in which the tension was controlled. The findings in these 13 eyes indicate that cyclodiathermy exerted its effect by reducing the flow of aqueous into the eye.

COMMENT

One of us (H. G. S.) has previously reported the results obtained in 16 eyes operated on by the original Vogt technique

TABLE 3.—*Tabulation of Tonographic Studies on Thirteen Eyes Operated on by Cyclodiathermy*

Patient	Diagnosis	Date of Cyclodiathermy	Preop. Tension, Mm. Hg	Tonography Before Cyclodiathermy *		Postop. Tension, Mm. Hg	Tonography After Cyclodiathermy	
				C	*K*		*C*	*K*
E. L.	Chronic simple glaucoma OS	1/24/51	35	....	....	19	0.08	0.69
M. H.	Chronic simple glaucoma OS	3/ 7/51	48	....	....	48	0.05	2.57
D. B.	Glaucoma sec. to uveitis OS	4/25/51 9/ 6/51	60 28	....	....	57	0.04	1.74
H. S.	Aphakic glaucoma OS	6/ 6/51 10/24/51	36 45	....	....	..	0.13	2.96
A. N.	Chronic simple glaucoma OS	6/12/51 3/ 5/52	62 40	....	....	27	0.07	1.06
J. W.	Chronic simple glaucoma OD	9/12/51 2/13/52 2/20/53	36 50 35	....	....	40	0.04	1.07
A. W.	Chronic simple glaucoma OD	10/10/51	30	....	....	28	0.10	1.51
L. D.	Glaucoma sec. to uveitis OD	10/24/51	55	....	....	37	0.04	0.97
F. B.	Chronic simple glaucoma OS	2/20/52	55	....	....	44	0.10	3.09
M. L.	Glaucoma sec. to uveitis OD	10/ 1/53	80	....	....	16	0.14	0.81
T. B.	Aphakic glaucoma OS	5/ 6/54	60	0.06	1.68	20	0.11	1.01
M. J.	Glaucoma sec. to uveitis OS	5/13/54	57	0.04	1.53	8	0.15	0.02
M. D.	Glaucoma sec. to herpes zoster ophthalmicus OS	5/12/54	52	....	....	30	0.07	1.20

* *C* indicates facility of outflow; *K*, rate of aqueous production.

of cyclodiathermy. This type of operation was discontinued in 1949 because of the erratic control of tension and because placement of the cautery 3 mm. from the limbus resulted in a rather high incidence of corneal edema and opacification.

More recently, techniques have been developed which require only a few applications of the electrode at least 6 mm. behind the limbus.

Because of increasing enthusiasm for these methods in the literature, we began to employ an operation discovered by Reiser. Eight to 10 applications of a 1.0 to 1.5 mm. electrode are made through the intact conjunctiva 6 mm. behind the limbus over slightly more than half the globe.

Thirty-eight eyes were operated on by this method. Tension was controlled in 15 eyes without miotics and in 6 more by the supplementary use of miotics, while tension remained uncontrolled in 17 eyes. The operation was used in primary glaucoma when other operations had failed, or when the presence of residual fields made it undesirable to lower the tension abruptly, in infantile glaucoma, and in glaucoma secondary to uveitis and aphakia. Neither sympathetic ophthalmia nor any other serious complication was encountered.

It is apparent that the operation does not always succeed in controlling ocular tension. Results are almost uniformly good for a few months, but tension may again rise as much as six months, or even a year, after operation. It seems to offer one of the safest approaches to glaucoma, however, and we continue to advocate its use when more conventional surgery has failed, or when ordinary surgical methods are considered hazardous.

Reduction of the *K* value in all eyes in which the ocular tension has been controlled indicates that control of ocular tension results from a diminution in the production of aqueous humor. Increased ocular tension, therefore, is replaced by diminished aqueous production, an equally abnormal situation.

SUMMARY AND CONCLUSIONS

A recent method of cyclodiathermy involving 8 to 12 applications of the electrode placed 6 mm. or more back of the limbus for 10 seconds at each site proved to be a safe operative procedure in this series of 38 eyes.

Control of ocular tension is not uniformly successful. The tension was controlled in 15 eyes without miotics and in 6 more with miotics given postoperatively. Failure occurred in 17 eyes.

Tonographic studies indicate that reduction of intraocular tension was the result of marked inhibition of aqueous production.

We believe that the operation is indicated in primary glaucoma when other, more conventional, operations have failed or are considered hazardous, or in secondary glaucoma if operation cannot be avoided.

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Evaluation of the Sporicidal Activity of Post's Sterilizing Solution

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For many years ophthalmic surgeons have been utilizing germicidal solutions in attempts to sterilize their instruments. The fact that the cutting edges of the instruments are not injured by these solutions has popularized their use. The reliability as sterilizers has recently been questioned, however.

It is essential at the time of an operative procedure that the surgeon's instruments be as sharp as they were prior to sterilization. Autoclaving is an unequivocal method of achieving sterility and is adequate for the instruments used in general surgery. Unfortunately, however, autoclaving may cause some deterioration of the sharpness of the delicate instruments used in ophthalmic surgery. Another certain way of achieving sterilization is by means of dry heat. The disadvantage of this method is the length of time it takes, i. e., the time required to heat an oven to 160 C, plus the sterilizing time of two hours at that temperature. This is in general agreement with Allen's finding, and opposed to the reported work of Post.

A reliable sterilizing solution would have many obvious advantages over both these thermal techniques. A solution widely used for this purpose, the efficiency of which in destroying the vegetative forms of bacteria has been well established,[1] is the so-called "Post's solution #4."* Its formula is as follows:

Saponated cresol solution N. F.....	8%
Lavender oil.....	2%
Thymol crystals.....	2%
Ethyl alcohol 95%.....	88%

This solution has been relatively successful in the clinical trials to which it has been subjected. Recently, however, Post [2] and Allen,[3] working independently, reconsidered the question of its sterilizing ability with respect to spores. Their results were contradictory, and they both requested that further studies be made in a third laboratory. In response to this request, a bacteriologic evaluation of the solution was undertaken in this laboratory. As a preliminary test we found Post's solution to be bactericidal against Pseudomonas aeruginosa. This is in agreement with Post's statements [1] and with the general concept of alcohol as a bactericide, but in contrast to Allen's recent report.[3] However, its sporicidal activity is the subject of the present report.

In theory at least, the ideal way to evaluate a surgical germicide is to test the instruments actually used in an operative procedure. The conditions of such an experiment are so difficult to control, however, that significant results are rarely achieved. For practical purposes it is better to determine the type and extent of contamination on sample instruments and then duplicate the conditions as closely as possible in controlled laboratory tests.

This experimental work was done in the Heller and Proctor Laboratories as a joint project of the School of Pharmacy (Dr. Riegelman) and the Department of Ophthalmology (Dr. Vaughan and Mr. Okumoto), University of California, aided by grants from the University of California Research Committee.

* In this paper the terms "Post's solution" and "solution #4" are used interchangeably.

ANALYSIS OF TESTING PROCEDURE

Examination of the reports of the previous investigators reveals slight differences in their experimental techniques. Some of these differences were discussed by Post in a recent paper.[2] In all essential respects the procedures were the same, however, and for the purposes of this discussion are so considered. In our opinion this method was deficient from a bacteriologic standpoint. The Figure summarizes the principal steps taken by Post and Allen and the modified steps taken by us. All the elements of the original procedure were analyzed, as follows.

Post's Solution

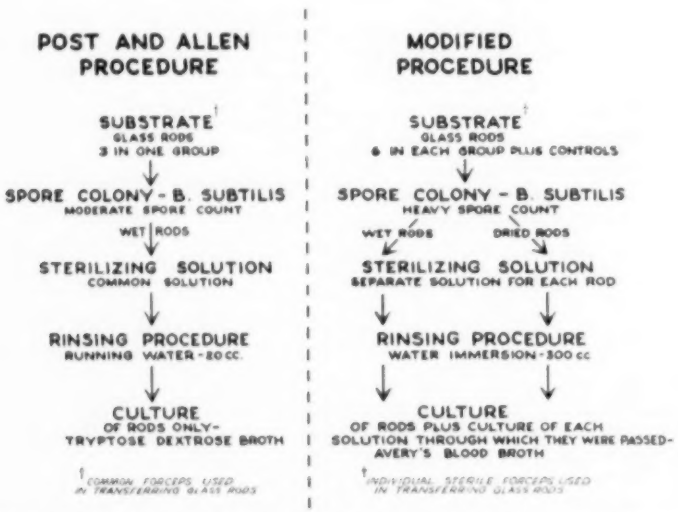
As indicated above, Post's solution contains a combination of the following known antibacterial agents:

1. *Cresol and Thymol.*—These phenolic derivatives are bactericidal even in the presence of extraneous organic matter, their activity closely paralleling that of phenol. They have little or no sporicidal activity.[4]

2. *Alcohol.*—Any strength of ethyl alcohol above 40% by volume is effective within five minutes against the common non-spore-forming pathogenic bacteria. Higher concentrations of 60% or 70% or more are effective when the organisms are in a dry state. Alcohol by itself has little effect against bacterial spores, however, and may even sustain their growth. In our experiments we tested only the original solution # 4, which contains ethyl alcohol 95%. Post recently suggested using isopropyl alcohol 91% instead, for reasons of economy. According to the literature, this compound is also an excellent bactericide and a poor sporicide.[5]

3. *Accessory Components.*—(*a*) Lavender oil was added because of the unpleasant odor of the mixture. This volatile oil possesses very little, if any, antibacterial action.

(*b*) Saponated cresol solution N. F. is essentially a dispersion of cresols in a weak alcoholic solution. From an analysis of the official formula, we may estimate that it also contains from 30% to 35% potassium soaps of the vegetable oil used in the preparation. This results in a 2.5% concentration of soap in the finished solution # 4. While soaps are recognized proteolytic agents with antibacterial activity, they are much less bactericidal in the presence of organic matter and appear to have little or no sporicidal activity under test conditions. However, a soap solution of this concentration is highly detersive and could wash away an aqueous suspension of



Comparison of the modified sporicidal test procedure with the former Post and Allen procedure.

spores. Another factor to be borne in mind is the much greater detersive action of soaps when used on glass than when used on metal.

Glass Rods

According to Post's procedure, a glass rod is passed through a Bacillus subtilis spore broth, thereby acquiring on its surface a thin aqueous suspension of spores. The rod is then transferred to solution #4. Since solution #4 contains powerful wetting agents, careful consideration must be given to the possibility that the mere washing of the spore film from the rods may be misinterpreted as sporicidal activity. Several experiments in which the wetting agents alone were used demonstrated clearly the ability of such a solution to wash from the rods a large majority of the spores without killing them.

An instrument which has been in contact with body fluids is coated with a layer of proteinaceous material, which coagulates upon standing and becomes a dry film. Any micro-organisms contained within this film are protected by a more or less continuous mechanical barrier. A procedure to test the reliability of a sterilizing solution should therefore include among its conditions the approximation of a bloody instrument that has been permitted to dry for several hours.[6] To provide such a condition, glass rods were passed through the B. subtilis spore broth and then air-dried in a sterilizing oven for 15 minutes at a temperature of 95 C.

Spore Colony

To test the sporicidal activity of a solution, the test broth must contain a good growth of spores. In recent experiments Post and Moor [2] used a new, and apparently more resistant, strain of B. subtilis, which they obtained from Dr. H. F. Allen. A sample culture of this organism was sent us by Dr. Allen. Unfortunately, in our laboratory this particular sample was low in spores, the vegetative cells far outnumbering them. For this reason we chose for our experiments another B. subtilis strain, which had a high spore count.

Forceps

In discussions of this experimental problem, much has been said † about the manner of handling the forceps that are used to pass the rods through the various stages of the procedure. In his original experiment, Post utilized two forceps for the entire procedure, immersing them in samples of solution #4 between each step. Allen, also using two forceps, attempted to sterilize them between steps of the procedure by flaming. Whether the difference in the results achieved by the two investigators was related to the manner of sterilizing the forceps is an unresolved question. To eliminate this variable, a large supply of sterile forceps was kept on hand and each forceps was used only once.

Rinsing Procedure

Post's procedure called for the pouring of some 20 cc. of water over the rods to flush off the residual solution #4. We elected to immerse each individual rod in 300 cc. of sterile distilled water and to subject each rod to a standard amount of agitation in this wash water.

Culture Media

A tryptose-dextrose broth was used for the test cultures in the original procedure. As a result of considerable study, this culture medium was regarded as unsatisfactory for growing spores. When inoculated with viable spores, it produced poor growth, and occasionally none at all. Reddish [7] has pointed out that spores may be fastidious in their growth requirements and that proper nutrient media are necessary to test for their presence. In our preliminary experiments we used both thioglycollate and Avery's glucose broth containing defibrinated rabbit's blood. Although B. subtilis grew in thioglycollate broth, this medium had certain disadvantages in comparison with Avery's broth: (1) Growth was not uniformly dispersed in it, and (2) since a certain amount of precipitation and protein coagulation takes place when Post's solution is added to thioglycollate, it was occasionally

† References *2* and *3*.

difficult to differentiate chemical precipitation from bacterial growth. Avery's broth, on the other hand, proved to be an excellent culture medium for all the strains of B. subtilis investigated.

MODIFIED TESTING PROCEDURE

The testing procedure finally adopted for this study is summarized in the Figure and may be described as follows:

A total of 14 sterile rods were used for each test. Two of the 14 rods were immersed in a spore dispersion and cultured directly to verify the viability of the spores. The remaining 12 were divided into two groups of 6 rods each. The six rods of Group 1 were immersed in a spore dispersion and transferred immediately to separate Petri dishes containing solution # 4. The six rods of Group 2 were immersed in the spore dispersion and dried in a sterilizing oven for 15 minutes at 95 C; each rod was then placed in a separate Petri dish containing solution # 4.

After a 10-minute exposure to solution # 4, each of the 12 rods was placed in 300 cc. of sterile distilled water, agitated uniformly for 2 minutes, and transferred to Avery's broth. A loopful of solution # 4 from each Petri dish, and 1 cc. of wash water from each container, were cultured simultaneously in samples of Avery's broth.

In a second test, in which the same procedure was followed, the rods were immersed in solution # 4 for a 30-minute period.

RESULTS

The experimental results summarized in the Table show conclusively that the solution tested was not sporicidal. Not only were all the test-rod cultures positive that were run after the rod had passed through the system, but cultures of the samples of Post's solution in which the wet contaminated rods of Group 1 had been immersed and cultures of samples of the wash water in which they had been agitated were also positive. Cultures of the rods that had been dried after contamination (Group 2) were also positive, but in this case the cultures of 9 of the 12 samples of Post's solution in which they had been immersed were negative. The wash-water cultures, on the other hand, were again all positive, and from this it could be deduced that the alcohol-cresol solution itself was not an efficient detersive agent on dry proteinaceous film but that agitation in large quantities of wash water consistently removed enough of the adherent spores to give positive cultures.

Bacteriologic Evaluation of Post's Sterilizing Solution #4

Minutes' Exposure	Sol.-4 Culture	Wash-Water Culture	Test-Rod Culture
Wet rods			
10	6 positive	6 positive	6 positive
30	6 positive	6 positive	6 positive
Dried rods			
10	1 positive 5 negative	6 positive	6 positive
30	2 positive 4 negative	6 positive	6 positive

COMMENT

It is indeed fortunate that infection with spore-forming organisms as a complication of ocular surgery is extremely rare. Such infection is characteristically very severe, however, and occurs often enough to constitute a real danger. The spore-killing potential of a sterilizing solution, moreover, is a highly satisfactory criterion of the solution's reliability, since sporicidal efficiency guarantees that all vegetative forms will have been destroyed.

On this basis, it seems clear from an analysis of the ingredients of Post's solution, and, more importantly, from the results of the testing procedure recorded above, that this solution is open to question as an adequate sterilizing medium. The bacteriologic results summarized in the Table indicate that if a few spores remained on the surface of a well-cleaned surgical instrument, they could easily pass through this entire cold sterilizing system without being affected. If the instrument had not been properly cleaned and contained a few spores in an aggregated protein film, the chances for sterilization in this system would probably be quite remote.

The fact that the results reported herewith differed from those of the previous workers can probably be accounted for by the differences in experimental procedure, i. e., the use of Avery's blood broth as a selective culture medium for B. subtilis spores, the availability of a large number of sterile forceps so

that each forceps was used but once, the use of a separate Petri dish containing Post's solution for each rod, and the use of larger amounts of water to wash the rods. The culturing of each solution through which the rods passed yielded the interesting information that Post's solution itself could sustain B. subtilis spores, and that it was not an efficient detersive agent on dry proteinaceous film. In this connection it should be noted that in reporting his tests on glass rods contaminated with B. subtilis, Post has stated repeatedly that the rods were apparently sterile when cultured at the end of his procedure; he has at no time reported attempts to recover spores at any intermediate stage of the testing procedure.

The use of rods dried after contamination as a more stringent testing device than wet rods was rewarding and suggested that no procedure for the evaluation of a chemical sterilizing solution should be limited to tests on wet instruments alone. It is recommended that test spores be dried on test instruments as a first step in any procedure designed to evaluate the sporicidal potency of a chemical solution to be used for sterilizing ophthalmic surgical instruments.

SUMMARY AND CONCLUSION

An investigation of the sporicidal activity of Post's solution, a widely used sterilizing medium for ophthalmic surgical instruments, is reported.

When evaluated according to a modification of the testing procedure followed by Post and Allen, the solution was found to be non-sporicidal.

A procedure to test the reliability of a solution designed for the sterilizing of surgical instruments should include among its conditions the approximation of an instrument upon which blood has been allowed to dry. It is therefore recommended that test spores be dried on test instruments as a first step in any procedure used to evaluate the sporicidal potency of a solution.

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Ocular Findings in Cerebral Palsy

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Cerebral palsy is a neuromuscular disability resulting from an injury to the motor centers of the brain. The condition was first described by Little in a monograph published in 1843.[1] In the past decade it has been brought to social and medical consciousness and characterized by the work of such men as Phelps,[2] Perlstein,[3] Crothers,[4] and Deaver.*

While much has been written on the general condition, Guibor † is probably the only contributor to the ophthalmic literature. He noted that over 50% of patients with cerebral palsy have oculomotor anomalies, the commonest being defects of horizontal gaze and esotropia. Conservative therapy with atropine, lenses, and prisms was described. Guibor stressed that small children have a remarkable capacity for recovery from motor anomalies when treatment is instituted at an early stage. This observation is borne out by the experimental work of Kennard [9] on young monkeys.

The purpose of the present paper is to form a concept of the ocular manifestations of cerebral palsy and to arrive at a method of management.

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* Deaver, G. G.: Personal communication to the author, Nov. 15, 1954.

† Reference 5 through 8.

CLASSIFICATION

According to the lesions in the motor centers of the brain, cerebral palsy may be divided into three major groups: namely, (1) spastic, with lesions in the cerebral cortex or pyramidal tracts; (2) dyskinesic, with lesions in the basal ganglia, and (3) ataxic, with lesions in the cerebellum. This classification is based on clinical findings, and, in general, 95% of the cases are diagnosed as one of these distinct types.

ETIOLOGY

Cerebral palsy may be congenital, due to hereditary causes, prenatal influences, and birth trauma, or acquired, due to postnasal causes, such as trauma, infections, vascular accidents, and neoplasms.

INCIDENCE

The incidence of cerebral palsy is difficult to determine, and surveys usually exceed the anticipated occurrence. Phelps [2] estimated that it occurs 6 times per 100,000 population, resulting in approximately 200,000 cases up to the age of 20 years. Thus, it is second only to poliomyelitis as a crippler of young people in the United States today.

A New York State survey [10] indicated that cerebral palsy occurs about 6 times in every 1000 births.

MATERIAL AND METHODS

One hundred unselected cases from the cerebral palsy clinic at Lenox Hill Hospital were examined and followed for two years. Fifty-seven per cent were spastic, 34% dyskinesic, and 9% ataxic. All were congenital in origin. Since this distribution approximates the usual partitioning of any cerebral palsy group, this series of cases may be considered as representative.

TABLE 1.—*Ocular Findings in One Hundred Cases of Cerebral Palsy*

Normal findings	44%	
Esotropia	40%	
Exotropia	8%	
Paresis of upward gaze	1%	
Optic atrophy	3%	56%
Congenital cataracts	2%	
Coloboma of iris	1%	
Spastic eyelids	1%	
	100%	
Concurrent conditions		
Nystagmus	2 cases	
Papilledema	1 case	

The eye examination included visual acuity determination, cycloplegic refraction, visual field estimates, ophthalmoscopy, and, especially, muscle balance studies.

The ocular findings are tabulated in Table 1.

RESULTS

Forty-four per cent of the patients had normal ocular findings, while 56% had abnormal findings. When these figures are compared with a cerebral palsy incidence of 6 per 1000 live births, it can be estimated that 3.3 per 1000, or 1 per 300, live births will have cerebral palsy and some ocular abnormality. Perlstein [3] stated that any child with eye abnormalities at birth must be considered to have brain damage until it is proved otherwise and deserves a complete neurologic examination.

Abnormalities of muscle balance are most prominent, 40% of the group showing esotropia and 8% exotropia. The cases of optic atrophy (three), congenital cataract (two), and coloboma of the iris (one) may be grouped together as instances of developmental defects, giving an incidence of 6%. Gaze palsy was found in one case and spastic eyelids in another. Nystagmus occurred in two cases and papilledema in one.

Table 2 shows the distribution of cases of normal and abnormal ocular findings according to the three cerebral palsy groups.

It is apparent that the eye manifestations of cerebral palsy give no clue as to whether a patient is spastic, dyskinesic, or ataxic, because any of the conditions may be found in the three groups. Furthermore, the eye condition cannot be anticipated on the basis of the clinical classification (i. e., spastic, dyskinesic, or ataxic). It must be assumed that the brain damage is diffuse and seldom localized to one specific area of the brain. This assumption is borne out by the work of Josephy,[10] whose postmortem studies indicate little or no correlation between the clinical picture and the brain damage as noted microscopically.

COMMENT

With no guide to the proper ophthalmologic management of these patients, the experience of physicians specializing in the treatment of cerebral palsy was utilized. According to Phelps,[2] spastic patients respond well to surgery of antagonist muscle groups, muscle transplantation, and neurectomy, while dyskinesic and ataxic patients respond to physical training.

With these facts in mind, generally accepted ophthalmologic methods of treatment were followed. In the field of oculomotor imbalance, for example, atropine refraction, lenses, occlusion, surgery, and exercises were prescribed. I did not administer neostigmine (Prostigmin), useful in relieving spasm, for, according to Perlstein,[11] the drug causes psychologic changes if continued for the length of time necessary to be effective

TABLE 2.—*Distribution of Ocular Findings Among Cerebral Palsy Groups*

Ocular Findings	Spastic	Dyskinesic	Ataxic	Total
Normal findings	23 (40.4%)	18 (52.9%)	3 (33.3%)	44
Esotropia	25	13	2	40
Exotropia	7	0	1	8
Paresis of upward gaze	0	1	0	1
Optic atrophy	1 (59.6%)	1 (47.1%)	1 (66.6%)	3
Congenital cataract	0	1	1	2
Coloboma of iris	0	0	1	1
Spastic eyelids	1	0	0	1
Total	57	34	9	100

in training. Guibor,‡ however, found neostigmine to be efficacious in cases of paralysis of the levator associated with ptosis when the drug was administered once or twice daily.

Esotropia.—Of 40 patients with esotropia, 15 had sufficient accommodative elements to warrant the use of their cycloplegic acceptance. As little as +2.00 D. sphere was helpful in certain patients. Twelve cases became alternators after occlusion by atropine and patching. It the patient was seen early enough, visual acuity was maintained, or improved, and few cases of amblyopia resulted.

Surgery was performed on seven patients. Six had excellent cosmetic and functional results. One patient, operated upon at another hospital, was overcorrected and is now exotropic. Surgery is contemplated in four other patients. Of the seven patients operated upon, three were dyskinesic and four were spastic. While this is an extremely small number from which to draw conclusions, it seems feasible to operate on patients in the dyskinesic group, as well as the spastic group, provided the angle of deviation remains relatively constant. No experience was obtained with ataxic patients. Three patients were myopic and were given glasses. Three others were mentally retarded to such a degree that therapy was thought inadvisable. It must be noted that there is some overlapping in this group, for many cases were treated by more than one method (i. e., glasses, occlusion, or surgery).

Nine patients who had esotropia received no specific treatment for the ocular condition, but fusion improved and the esotropia decreased. They were not confined to any one group. These results may be explained by the fact that the brain is the center of many functions, one being ocular.§ A cerebral palsy child needs treatment in all spheres (ocular, muscular, hearing, speech, etc.). Deaver § has noted that as one area improves, others improve. He has stressed that since rehabilitation often depends on good visual acuity, early ophthalmic treatment is essential.

Exotropia.—Of eight patients with exotropia, three had no significant refractive error, and surgery is contemplated in the future. One myopic patient in this group achieved a good result with lenses and exercises to improve convergence. One patient had a convergence paralysis and exotropia, while another had progressively decreasing exotropia without treatment directed toward the eye condition. Two patients were so mentally retarded that therapy was considered inadvisable.

Among the 48 cases of muscle imbalance, 10 children were improved without specific ocular care. During brief moments of complete relaxation, these children could achieve binocular fusion. Apparently, as the binocular experience increased, the stimulus to fusion was enhanced, and they learned to overcome their original defect.

Orthoptic treatment played a limited role in this study, owing to limitations of time and money. This therapy was curtailed because the children were so completely occupied with other training programs. It is believed that it could be of value if properly and skillfully supervised.

Paralysis of Upward Gaze.—One patient with paresis of upward gaze was encountered.

Optic Atrophy.—Of the three cases of optic atrophy, two were associated with severe mental retardation and cerebral dysfunction. The optic atrophy was attributed to cerebral agenesis. The third case was secondary and followed hydrocephalus.

Congenital Cataracts.—The two patients who had congenital cataracts were operated upon monocularly. One achieved 20/70 vision, while the other has had one needling operation and may require another.

Coloboma of Iris.—The coloboma of the iris, observed in one patient, was strictly a development anomaly. When this case is grouped with three cases of optic atrophy

‡ Guibor, G.: Personal communcation to the author, Oct. 26, 1954.

§ Personal communication to the author, Nov. 15, 1954.

and two cases of congenital cataracts, the incidence of developmental defects is 6% in this series. This is higher than the expectancy in a normal population, i. e., 0.6%,[12] and bears out the observation of Yannet.[13] He reported the occurrence of congenital ocular malformations in 15% of 99 cases of cerebral palsy.

Spastic Eyelids.—Spasticity of the eyelids was observed in one child. When he attempted to close his eyes, the eyeball would roll upward but the palpebral fissures would not close. They closed only when he was sound asleep. The family could always tell when he was feigning sleep because the white sclera would be exposed.

Concurrent Conditions.—Although it was anticipated that nystagmus might be a frequent finding, only two cases of nystagmus were observed, and neither was associated with gaze palsy. One case was of the dyskinesic and the other of the spastic type. Both showed marked spontaneous improvement.

One case of papilledema occurred in a child who died of a massive cerebral astrocytoma, the presence of which was considered incidental to the cerebral palsy.

It is interesting to note that no gross visual field defects were found. Tizard, Paine, and Crothers,[14] using the same testing methods as those employed in this study (i. e., confrontation), found homonymous hemianopsia in 25% of 106 cases of cerebral palsy.

SUMMARY

In this series of 100 unselected patients with congenital cerebral palsy the following observations were made:

1. Fifty-six per cent showed ocular defects, while 44% revealed no ocular abnormalities.

2. The distribution of normal and abnormal ocular findings among the three cerebral palsy groups (namely, spastic, dyskinesic, and ataxic) indicated that the various defects could occur in any of the three groups. It is impossible, therefore, to anticipate what the ocular condition will be merely on the basis of the clinical classification of cerebral palsy.

3. Abnormalities of muscle balance were most prominent, with 40% showing esotropia and 8% exotropia.

4. In the management of esotropia, as slight a correction as a +2.00 D. sphere was useful in relieving accommodative squint. Occlusion was an extremely useful tool in preventing amblyopia and inducing alternation. There appeared to be no contraindication to surgery in the spastic and dyskinesic groups provided the angle of deviation was constant. No surgical experience was obtained in the ataxic group.

5. Ten patients improved and overcame their muscular imbalance during the course of training in other spheres. This may have been related to increasing binocular experience. On the other hand, improvement in the ocular status frequently aided rehabilitation of the total patient.

6. Developmental ocular abnormalities occurred in 6% of this cerebral palsy group, or 10 times as often as in the general population.

7. Gaze palsy (one case) and nystagmus (two cases) represented a very small part of this series. No gross visual field defects were found.

8. Atropine was used successfully to relieve accommadative spasm and as an adjunct in patching to produce alternate fixation, thus preserving vision. Neostigmine (Prostigmin) was not used.

CONCLUSIONS

Cerebral palsy, a neuromuscular disability, affects 200,000 people up to the age of 20 years in the United States. The general state has been carefully described, but little has been written about the ocular manifestations.

On the basis of a clinical study of 100 patients with congenital cerebral palsy, 56% revealed ocular abnormalities, the greatest part of which were oculomotor imbalances. Other ocular involvement in this study included optic atrophy, congenital cataract, coloboma of the iris, gaze paresis, and spastic eyelids. Approximately 1 child in 300 live births will have cerebral palsy and some oc-

ular manifestations. Any child with eye abnormalities should have a careful neurologic examination to rule out cerebral palsy.

Generally accepted principles for the management of heterotropias can be applied. Relatively low plus spheres usually relieve accommodative squints significantly. There is no contraindication to surgery provided the deviation is relatively constant.

A certain number of heterotropias improve spontaneously as binocular experience increases. Nystagmus and visual field defects do not present a serious problem.

Cerebral palsy victims in our population represent a very significant group. Their ocular status has been largely ignored because of more obvious and limiting defects. However, every victim of this disease deserves an early eye examination. Careful diagnosis and adherence to accepted methods of therapy yield gratifying results.

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Keratoacanthoma of the Ocular Adnexa

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It is our purpose to describe the occurrence on the eyelid of an important benign tumor which is often mistaken for squamous-cell carcinoma and which has not received attention in the ophthalmic literature. This tumor, keratoacanthoma, was established as a clinical entity by MacCormac and Scarff [1] in 1936, and since then their observations have been substantiated by others.

The tumor is important because histologically it resembles a low-grade squamous-cell carcinoma and most commonly is diagnosed and treated as a malignant tumor. Judging from the available literature, the dermatologists appear to be the only group of medical practitioners who are generally aware of its existence. Yet its incidence is relatively high, as indicated by reports of as many as 76 cases in one single series.* Among other terms, the tumor has been referred to as molluscum sebaceum, because of a superficial resemblance to molluscum contagiosum, and as molluscum pseudocarcinomatosum; however, the term keratoacanthoma has become the most accepted.[4]

Keratoacanthoma most frequently appears as a discrete, solitary lesion on the skin of the face but may occur as a single or multiple lesion in other areas.[5] The tumor begins as a small papillary or warty lesion and enlarges rapidly, usually reaching maximum size in four to eight weeks. When it is fully developed, its base is 1 to 2 cm. wide, and it is elevated several millimeters. The surface is roughened, and usually umbilicated, with a central keratin core. The color is variable and ranges from the color of the skin to a distinct pink, which occurs from either neovascularization or inflammatory hyperemia.[6]

Histologic sections of these tumors show irregular masses of epidermal cells which tend to proliferate toward, but do not actually invade, the underlying corium. A characteristic feature is a crater filled with keratin, resembling a cutaneous horn, which presents through the epithelial surface. Acanthosis is regular and orderly, but some prickle cells are atypical with hyperchromatic nuclei. There is a tendency toward pearl formation and an increased number of mitotic figures. The dermal-epidermal border is infiltrated by cells of inflammation. Most commonly these are lymphocytes; however, eosinophiles, plasma cells, and sometimes polymorphonuclear leucocytes may also appear. The tumor is relatively avascular, but the surrounding blood vessels are dilated and somewhat engorged. At the peak of development, the tumor resembles a low-grade squamous-cell carcinoma histologically and usually is diagnosed as such by general pathologists.[6]

Finley cites Rook and Whimster's [7] differentiation of keratoacanthoma from squamous-cell carcinoma by the following criteria: 1. Keratoacanthomas arise from areas of normal skin showing no evidence of precancerous change. 2. The epithelial hyperplasia appears to affect mainly the deep normal structure, hardly affects the surface epithelium, and undergoes no spontaneous ulceration. 3. The degree of atypicality of epithelial cells in this type of lesion is always

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* References 2 and 3.

Fig. 1.—Clinical appearance of lesion on the lid.

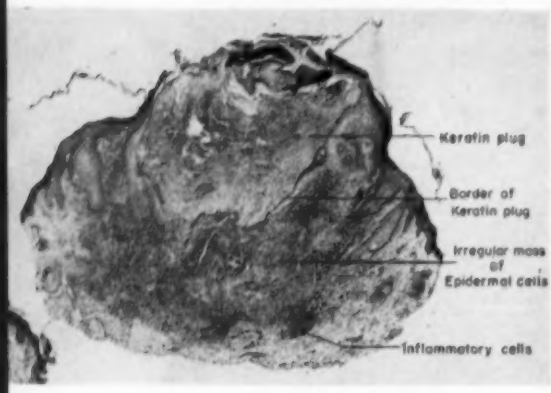
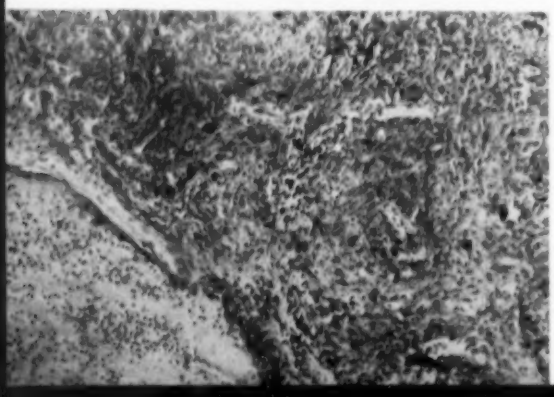


Fig. 2.—Low-power view of a microscopic section through the center of the lesion.

small. 4. One does not find true invading proliferation in this tumor.[2]

Clinically, the most significant characteristic is the rapid growth of the tumor to its maximum size in six to eight weeks.[7] If left undisturbed, keratoacanthoma will involute spontaneously with minimal scarring. The only residual may consist of a single depression of the skin with overhanging edges or a slightly thickened keratotic scar; however, no blemish may remain. A biopsy specimen of the tumor must generally be taken or the growth excised for diagnosis. If the true nature of the lesion is recognized, no further therapy is necessary. Roentgen therapy has been advocated also.†

Fig. 3.—High-power view of a section through an irregular mass of epidermal cells. It is this area that simulates low-grade squamous-cell carcinoma.



REPORT OF A CASE

In October, 1954, a 33-year-old white male physician first observed a rapidly developing papilloma along the outer lower lid margin, present for four weeks prior to consultation. He stated that growth of the tumor was rapid and enlargement was regular. There was no associated pain, tenderness, or discharge. There was no history of injury or of papillomas appearing elsewhere on his body.

Examination showed a discrete, firm, elevated papule arising from the skin on the inferior temporal aspect of the right lower lid (Fig. 1). There were superficial vessels extending up along the side of the tumor and disappearing into the epidermis at the apex. The surface was rough and umbilicated. The mass was painless on palpation and did not bleed on manipulation.

A clinical diagnosis of molluscum contagiosum was made, and an excision was performed. The pathologist's report was squamous-cell carcinoma of the lid, Grade 1. The presence of a large central keratin plug lying within a superficial crater of the skin constituted a striking feature of the microscopic sections. The anterior surface of this plug was exposed (Fig. 2), and surrounding it was a layer of hyperplastic epithelium, in which there was irregularity in cellular arrangement and size (Fig. 3). There was a tendency toward pearl formation, but no true pearls were noted. Acanthosis was evident, and although the tumor gave the appearance of invasion, actual extension into the corium was limited. Between the layer of epithelial hyperplasia and the underlying corium was an infiltration of inflammatory cells, predominantly lymphocytes.

Since excision, there has been no indication of recurrence. A fine, almost undetectable, linear scar remains.

SUMMARY

Keratoacanthoma is a tumor of the skin that may appear on the ocular adnexa. It is important because it is an innocent lesion with an excellent prognosis which, owing to its histologic resemblance to squamous-cell carcinoma, is usually confused with carcinoma, both clinically and pathologically. Although only recently recognized and foreign to ophthalmologic literature, this tumor is undoubtedly commoner than is generally recognized.

† References 8, 9, and 10.

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Sterilization of Tonometers

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Tonometry, the most important examination in the diagnosis of glaucoma, ought to be a perfectly safe one. Even a carefully performed tonometry sometimes results in minor abrasions of the corneal epithelium because of movements of the patient's eye or the tonometer, and keratitis may result in virulent micro-organisms from the tonometer are introduced into the corneal wound.

It is an established principle in ophthalmology that all solutions and instruments used by the eye surgeon on the patient's eye should be sterile, but most ophthalmologists sterilize their tonometers only rarely. Some ophthalmologists who have been lucky enough to avoid tonometer infections for years may consider regular sterilization of this instrument an unnecessary precaution, a waste of time.

The risk of nonsterile eye droppers is now generally recognized, but it is difficult to understand that more attention has not been paid to the sterility of tonometers.

No micro-organisms can grow on the tonometer except in the thin film of fluid transferred from the surface of the cornea to its footplate. In an ordinary office, where the interval between tonometries may be a half-hour or more, this tear film on the tonometer evaporates, and some of the micro-organisms will probably die before the tonometer is used on the next patient. The situation is different in big clinics, where tensions are taken on patients in rapid succession. Here it may well happen that the tear film on the tonometer does not dry between the measurements and micro-organisms may survive and be transferred to the next patient.

Fortunately, eye infections transferred from one patient to another by the tonometer are not common; but they do happen, and undoubtedly they occur more frequently than is indicated by reports in the literature.

REPORTS OF TONOMETER INFECTIONS

When tonometer infections do occur, a number of patients are likely to become infected before the mode of transmission is discovered. Such outbreaks have been reported by Thygeson and Stone[1] (inclusion conjunctivitis) and by Thygeson[2] (epidemic keratoconjunctivitis). Thygeson advises sterilization of all instruments used on patients, including tonometers and contact lenses.

A recent study of epidemic keratoconjunctivitis spread by tonometry was published in 1953 by Cockburn, Nitowsky, Robison, and Cheever.[3] The infection occurred among patients attending a glaucoma clinic. During a two-month period 9 out of 40 patients became infected.

Investigation revealed a number of possible modes of transmission, such as the fingers of the physicians, the eye droppers, various instruments, towels, and the tonometer. Only the tonometer was considered to be the likely vehicle of transmission, for only patients who had tonometry became infected. One tonometer was used for all patients examined for

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glaucoma and was "sterilized" after use only by swabbing with alcohol.

Further infections were prevented by the adoption of a more efficient method of sterilizing the tonometer. (This method is not described in the paper.)

USUAL METHODS OF TONOMETER STERILIZATION

Most of the methods described utilize solutions of various chemicals, with which the footplate of the tonometer is swabbed or in which it is immersed. These solutions include alcohol, ether, and benzalkonium (Zephiran).

In the directions for the use of Schiøtz tonometers the following advice is given: Before each use, the footplate should be wiped with cotton barely moist with alcohol, then wiped dry with sterile cotton.

Tower [4] recommends boiling the foot of the tonometer in an electric instrument sterilizer for five minutes after each tonometry and warns against the use of alcohol, ether, and benzalkonium as impractical and unsafe measures.

FLAMING OF THE TONOMETER

There is no doubt that boiling can sterilize the tonometer, but the method recommended by Tower is not simple or suitable as a routine office procedure. The most rapid method of killing micro-organisms is by flaming. Flaming of the platinum needle is an old procedure in bacteriology, and many ophthalmologists have occasionally sterilized their tonometers in the same way.

The fear is widespread, however, that flaming the tonometer will damage the expensive instrument, and nowhere in the literature have I found any information as to the length of time one has to hold the footplate of the tonometer in the flame to ensure efficient sterilization. It is also well known that the temperature of a flame is not uniform throughout, and it is difficult manually to hold the tonometer at a certain level in the flame.

A NEW INSTRUMENT

The purpose of this paper is to advocate sterilization of the tonometer after each use by means of an alcohol lamp which is constructed in such a way that it serves at the same time as a convenient tonometer holder and as a tonometer sterilizer (Fig. 1).*

Fig. 1.—Tonometer sterilizer.

The sterilizer is made of metal and consists of the following parts:

1. An alcohol container with a wick and a cap which can be screwed tightly on the container.
2. A vertical rod which fits into a sleeve on the side of the container.
3. A horizontal plate with a hole for the tonometer and a screw by which the height of the tonometer holder can be adjusted.

* Manufactured by E. B. Meyrowitz Surgical Instruments Co., Inc., 520 Fifth Ave., New York 36.

Only pure ethyl or methyl alcohol should be used, as alcohol leaves practically no deposit on the tonometer. After the tonometer has been sterilized many times between tonometries, a slight yellowish deposit may be seen on the footplate, probably consisting of inorganic substances (salt) from the tears, but this is easily wiped off with a piece of gauze or paper. When the tonometer is flamed frequently, there is no tendency for the plunger to become sticky, and cleaning of the cylinder will rarely become necessary. All organic substances are removed by burning.

During the sterilization the footplate should be at a constant level in the flame. Before the tonometer is moved into the flame, the height of the holder is adjusted so that the footplate is at a level between the upper and the middle third of the visible flame.

When these requirements have been fulfilled, the temperature which develops in the footplate of the tonometer during flaming will be dependent upon the number of seconds it is in the flame.

EXPERIMENTAL STUDY

The purposes of the laboratory studies reported here are (1) to learn how many seconds it is necessary to flame the tonometer footplate under the conditions described above in order to kill four common nonspore-bearing organisms and a spore-bearing one (Bacillus subtilis); (2) to find what temperature is developed in the footplate of the tonometer by flaming it for a certain number of seconds.

The technique of contaminating the tonometer and the technique of transferring the organisms from the tonometer to the agar surface approach the way in which a tonometer is used in practice.

A series of identical control experiments are performed without flaming the tonometer.

METHOD

Forty-eight-hour subcultures of organisms are used. In the case of B. subtilis cultures are grown five to six days to ensure maximum spore formation. The cultures are grown in meat infusion broth.

A Schiøtz X-tonometer of the most recent model was used in all experiments. The lower parts of this tonometer are made of stainless steel.

By the following method, the tonometer is contaminated by a thin film of fluid simulating the tear film on the cornea in vivo: The test block in the tonometer case ordinarily used for checking the zero point of the tonometer scale is sterilized by flaming and placed in a sterile Petri dish. A drop of a pure broth culture of the organism is transferred with a loop to the upper convex surface of the test block. The tonometer is contaminated by placing it on the test block for a moment. Then it is placed successively on 10 different areas of one-half of the surface of a sterile blood agar plate. The tonometer is not contaminated again between these 10 applications. This is the control experiment.

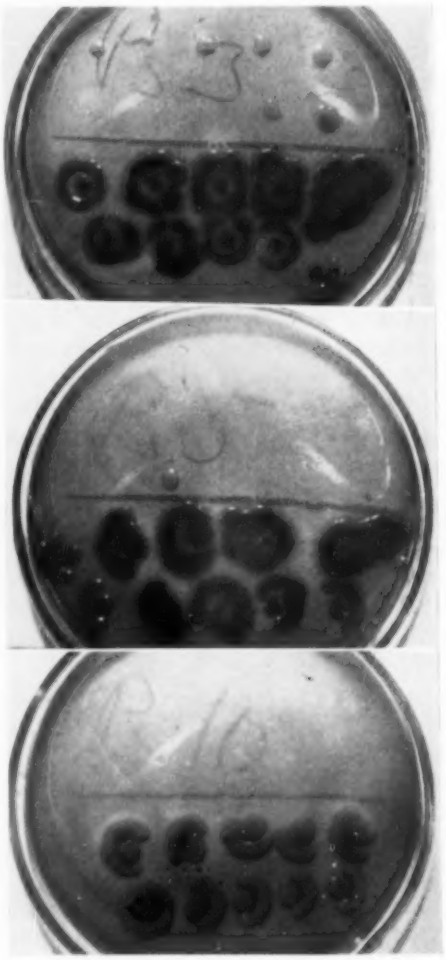

Fig. 2.—Tonometer sterilizing experiment with Pseudomonas aeruginosa. Abundant growth in all the controls. Flaming of the tonometer for three seconds is insufficient. Growth is seen in seven plunger imprints. Flaming of the tonometer for five seconds results in only one "plunger" colony. Flaming of the tonometer for 10 seconds: no growth.

The other half of the blood agar surface is used for the sterilization experiment. The circumference of the lower concave surface of the footplate leaves an imprint of a circle on the agar, and the plunger forms a small depression in the center of the circle.

A new loopful of the broth culture is now transferred to the surface of the test block, and the tonometer is again contaminated by placing it on the test block.

It is then put into position in the holder plate of the sterilizer. The tonometer holder has been adjusted to the proper height, so that the footplate is on a level with a plane between the upper and the middle third of the visible flame.

The contaminated tonometer is rotated into the flame and kept there a certain number of seconds, measured as exactly as possible with a stopwatch. Then it is moved out of the flame and allowed to cool off enough to avoid melting the agar. The tonometer is now again placed on 10 different areas on the other half of the blood agar surface in the same way as was previously done.

In different experiments, five organisms are used, and the tonometer heated from 2 to 15 seconds. In the case of the spore-containing B. subtilis culture the tonometer is flamed up to 40 seconds.

The cultures are incubated aerobically at 37 C and inspected after 24 and 48 hours. All experiments are repeated several times until the results are considered to be reproducible. As an example, three plates are shown in Figure 2 (copies of Kodachrome photographs).

RESULTS

In the Table the results are recorded as +10 if there was growth in all 10 imprints of the tonometer on the agar after 24 or 48 hours' incubation time in all the experiments. This was seen in all the controls.

The result is recorded as 0 if no growth was seen in any of the tonometer imprints. In the Table it is seen that heating the tonometer two seconds killed Streptococcus pyogenes but did not prevent growth of any of the other organisms tested in any of the tonometer imprints. Streptococci are known to be very susceptible to heating.

Results of Tonometer Sterilizing Experiments with Five Organisms

	Flaming Time, in Sec.							
Organism	2	3	5	10	15	20	30	40
S. aureus	+10	+	+	0	0	..	..	..
S. pyogenes	0	0	0	0	0	..	..	..
E. coli	+10	+	0	0	0	..	..	..
P. aeruginosa	+10	+	+	0	0	..	..	..
B. subtilis	+10	+10	+10	+10	+10	+10	+	0

The result is recorded as + if some of the tonometer imprints showed no growth and others showed growth, however small, in any of the series of experiments at a certain heating time.

From the Table it is evident that all four non-spore-bearing organisms tested are killed when the tonometer is flamed for 10 seconds in the sterilizer as described, but B. subtilis spores are not inhibited in their growth by heating the tonometer for 10 seconds. To kill B. subtilis spores 40 seconds of flaming is required.

It is interesting that the plunger must be flamed a longer time to become sterile than the circumference of the footplate (Fig. 2). During the flaming the plunger is located at a lower level and more centrally in the flame than the periphery of the footplate. The temperature is lower here.

TEMPERATURE DEVELOPED IN THE FOOTPLATE

An attempt is made to measure the temperature which is reached on the surface of the tonometer footplate by heating it a certain number of seconds. This experiment is of interest not only for theoretical reasons but also in the following discussion regarding the possibility of damaging the tonometer.

The product called Tempilaq† is convenient for this purpose. It consists of materials of calibrated melting points suspended in an inert, volatile, noninflammable vehicle covering the range from 113 to 2000 F. The Tempilaq in a bottle marked 400 F is used in the following way: The suspension is shaken to uniform consistency, and a thin smear is applied to the upper convex surface of the footplate of the tonometer, which is placed in the sterilizer. The suspension dries almost instantly to a dull mark. The tonometer is moved into the flame as described above, i. e., the footplate being between the upper and the middle third of the visible part of the flame. The time is taken with a stopwatch from the moment the tonometer is turned into the

† The Tempilaq was furnished by Tempil Corporation, 132 W. 22d St., New York 11.

flame until the Tempilaq liquefies, which occurs quite sharply, indicating that a temperature of 400 F has been reached.

By this method it is found that flaming of the tonometer for 10 seconds in the standardized way described raises the temperature on the upper surface of the footplate to approximately 300 F; 40 seconds of flaming raises the temperature to 500 F.

DOES FLAMING DAMAGE THE TONOMETER?

Before I began my experiments, I asked the Department of Metallurgy of Massachusetts Institute of Technology to examine my tonometer. It was my plan also to have my tonometer examined after I had performed several hundred flaming experiments—up to 500 F in some cases.

I also planned to have my tonometer examined by one of the tonometer testing stations before and after my experiments for possible change in the calibration of the instrument.

Prof. Carl F. Floe examined my tonometer and discussed the matter with me. He felt that the planned metallurgical tests were not indicated, as it is a well-known fact that heating stainless steel to the temperatures in question can in no way damage the metal. Furthermore, as heating produces no change in the structure of the metal, the calibration of the instrument cannot be changed either.

These considerations have been confirmed in practice. Dr. Trygve Gundersen, Dr. Edwin B. Dunphy, and Dr. Henry F. Allen have tried the sterilizer in their private practice. By regular use of the flaming sterilization no visible changes in their tonometers have occurred.

The method can be recommended for all tonometers made of stainless steel, and most of the tonometers on the market are made of this material.

As spore-bearing organisms are not usually found on the eye, 10 seconds of flaming appears to be an adequate protection. It is necessary to wait two minutes for the tonometer to cool off before it can safely be used after 10 seconds of heating.

The susceptibility of different viruses to heat varies greatly and is difficult to investigate by sterilization experiments. It may be presumed, however, that flaming the tonometer is effective against both bacterial and viral contamination of the instrument.

SUMMARY

An inexpensive tonometer sterilizer and tonometer stand is described.

Flaming the tonometer footplate under standardized conditions in this sterilizer kills non-spore-bearing bacteria in 10 seconds.

Flaming does not damage a tonometer made of stainless steel and does not change its calibration.

The sterilization is easy to perform by this method and takes practically no extra time to be included in the office routine.

Only a sterile tonometer should be used on patients' eyes.

Dr. Trygve Gundersen and Dr. Alice Marston gave helpful criticism in this study.

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A New Scleral Shortening Operation

Preliminary Report

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Since 1933, when Lindner [1] introduced the sclerectomy procedure devised by Müller [2] to retinal detachment surgery, there have been numerous modifications of scleral resection.

At the present time the many types of scleral shortening procedures can be classified in two groups: the penetrating, or full-thickness, scleral resection, and the lamellar scleral resection of a variable thickness of the outer part of the sclera and infolding of the remaining lamella, as described by Dellaporta.[3] In the latter group one might include the implantation of polyethylene tubing in a previously prepared lamellar scleral resection bed, as discussed by Schepens.*

Consideration of the different methods of scleral shortening disclosed a different approach which was thought to hold promise. This new shortening procedure entails folding the sclera outward, in contrast to the present operations of resecting a penetrating strip of sclera or the infolding of a lamella of sclera.

Experimental evaluation of scleral outfolding led to a new shortening operation which was thought to be technically safe, quick, and uncomplicated.

This study was supported in full by the Retinal Detachment Research Fund of the Institute of Ophthalmology of the Presbyterian Hospital.

From the Department of Ophthalmology of Columbia University College of Physicians and Surgeons, and the Institute of Ophthalmology of the Presbyterian Hospital.

* Schepens, C.: Symposium: Retinal Detachment, New York Academy of Medicine, Section of Ophthalmology, Jan. 3, 1955.

EXPERIMENTAL PROCEDURE

Preliminary studies, using freshly excised beef and rabbit eyes, were done to develop a procedure for creating an outward fold of the sclera.

Scleral fold operations were then performed on the left eye of 20 chinchilla rabbits, in an attempt to study the long-term effect of this procedure. Pigmented rabbits were selected so that the response of the pigment epithelial layer of the retina could be more accurately observed. These animals were anesthetized with intravenous pentobarbital (Nembutal) sodium. The general anesthesia was augmented by topical application of tetracaine (Pontocaine). Ten per cent phenylephrine (Neo-Synephrine) hydrochloride was instilled preoperatively in the eyes operated on, to insure an adequate view of the fundus at the completion of the operation. Postoperatively, after ophthalmoscopic examination, 1% atropine ointment and 0.5% oxytetracycline (Terramycin) ointment were instilled into the eyes operated on.

The operations were rated and the animals were distributed into four groups, so that each group was representative of the operative variations. The four groups were established as survival periods of 4 days, 10 days, 21 days, and 45 days. An operative record was made listing any complication and describing the postoperative fundus picture.

The animals were killed by means of pentobarbital sodium after the fundus was examined.

EXPERIMENTAL RESULTS

In considering the specific survival experimental results, it is necessary to define the method of operative evaluation. The procedures were rated as technically complicated or uncomplicated, according to the course of the operation. The common complication was hemorrhage, either from the choroid or from the emissary veins. Vitreous loss was occasionally encountered. When possible, two observers appraised each operation according to the following criteria:

1. Uncomplicated. No technical difficulties and minimal hemorrhage, either choroidal or from the emissary vessels.

2. Complicated. More than minimal hemorrhage from the choroid or emissary veins, or vitreous loss, or any combination of these accidents.

The results of the in vivo studies are presented under the immediate fundus changes, and the gross, as well as the ophthalmoscopic, changes in the four follow-up periods.

I. Immediate Postoperative Fundus Picture

Retinal detachment was the predominant change. This was somewhat longer than the scleral fold and extended from about the ora serrata to the equator. It was usually irregular in nature, but often a major ridge of detachment coincided with the area of sclera folded. Retinal hemorrhages could usually be accounted for in the operative protocol, and were the result either of injury to the emissary veins or of direct trauma to the choroid during placing of a suture.

II. Four-Day Survival Period

(*a*) *Gross Appearance.*—The scleral fold was intact in all instances, and the sutures were in place. The conjunctiva and episclera were adherent to the area of operation.

(*b*) *Ophthalmoscopic Changes.*—The fundus picture varied little from that seen at the completion of the operation. Occasionally, choroidal hemorrhages were seen which had not been recorded previously. In several cases the retinal detachment took on a more solid appearance, suggestive of an exudative detachment. Intraocular hemorrhages, observed ophthalmoscopically, seemed to correspond with complications described in the record of the operation; injury to the emissary veins gave rise to more serious hemorrhage.

III. Ten-Day Survival Period

(*a*) *Gross Appearance.*—The scleral fold was intact in all instances. The episclera and conjunctiva were firmly adherent to the area of operation.

(*b*) *Ophthalmoscopic Changes.*—In the uncomplicated operations there was beginning reattachment of the retina. A small ridge of detached retina with minimal pigment proliferation and little or no choroidal hemorrhage was observed at the site of operation. In complicated procedures hemorrhages were common and there appeared to be more pigment proliferation. In one eye a subretinal hemorrhage seen immediately after operation had extended to the vitreous.

IV. Twenty-One Day Survival Period

(*a*) *Gross Appearance.*—In most eyes a scleral fold was present. However, in about one-third of the eyes the medial sutures had stretched or given way and the central part of the fold had flattened.

(*b*) *Ophthalmoscopic Changes.*—In the group classified as uncomplicated, only a small retinal fold or complete reattachment of the retina was observed. Minimal pigment reaction was present. In the eyes evaluated as complicated, signs of choroidal hemorrhage persisted, vitreous hemorrhage was circumscribed, and pigment proliferation was more extensive.

V. Forty-Five Day Survival Period

(*a*) *Gross Appearance.*—The scleral fold had flattened, but the site of operation was easily identified by the scar formation. The suture tracts could be recognized by small areas of transparency in the areas of scleral scar.

(*b*) *Ophthalmoscopic Changes.*—There was remarkably little change in the fundus at this time. The eyes which were rated as uncomplicated showed the retina in place; thin lines of pigment with a striated appearance signified the area of operation. In cases in which complications had occurred, the retina was also in place, but vitreous bands and occasional choroidal scars remained. In one eye graded as uncomplicated, a previously observed retinal hemorrhage had given rise to a vitreous band.

The accompanying Table attempts to correlate the complications observed ophthalmoscopically at the time the animal killed with operative ratings and the fundus observation following the procedure. Two arrivals died and were not included. The results indicate that the description recorded

at the termination of the experiment in each case accurately reflect the complications recorded in the operative protocol.

EXPERIMENTAL CONCLUSIONS

Inasmuch as human sclera is technically easier to operate on than the sclera of rabbit, it was concluded that the results of these animal studies justified a clinical trial of this operation.

CLINICAL APPLICATION OF EXPERIMENTAL FINDINGS

We have performed scleral folding procedures in selected cases of retinal detachments for which a shortening of the scleral coat was indicated. These cases have been ones which were prognostically poor with any type of operation. The operative technique resulted from the experimental studies and findings enumerated, with one exception. Absorbable surgical (gut) suture was used in the animal studies, but 4-0 black nonabsorbable (silk) suture material is being used at present because of the tendency of the gut suture to give way.

Summary of Experiments According to Complications

		Complications			Fundus Changes Immediately Postoperative			Fundus Changes at Time of Killing		
Operative Ratings	No. Rated	Choroid Hemorrhage	Emissary Hemorrhage	Vitreous Loss	Choroid Hemorrhage	Retinal Hemorrhage	Vitreous Hemorrhage	Choroid Hemorrhage	Retinal Hemorrhage	Vitreous Hemorrhage
Uncomplicated	9	3	1	0	1	0	0	1	0	1
Complicated	9	2	7	6	6	5	0	1	0	6

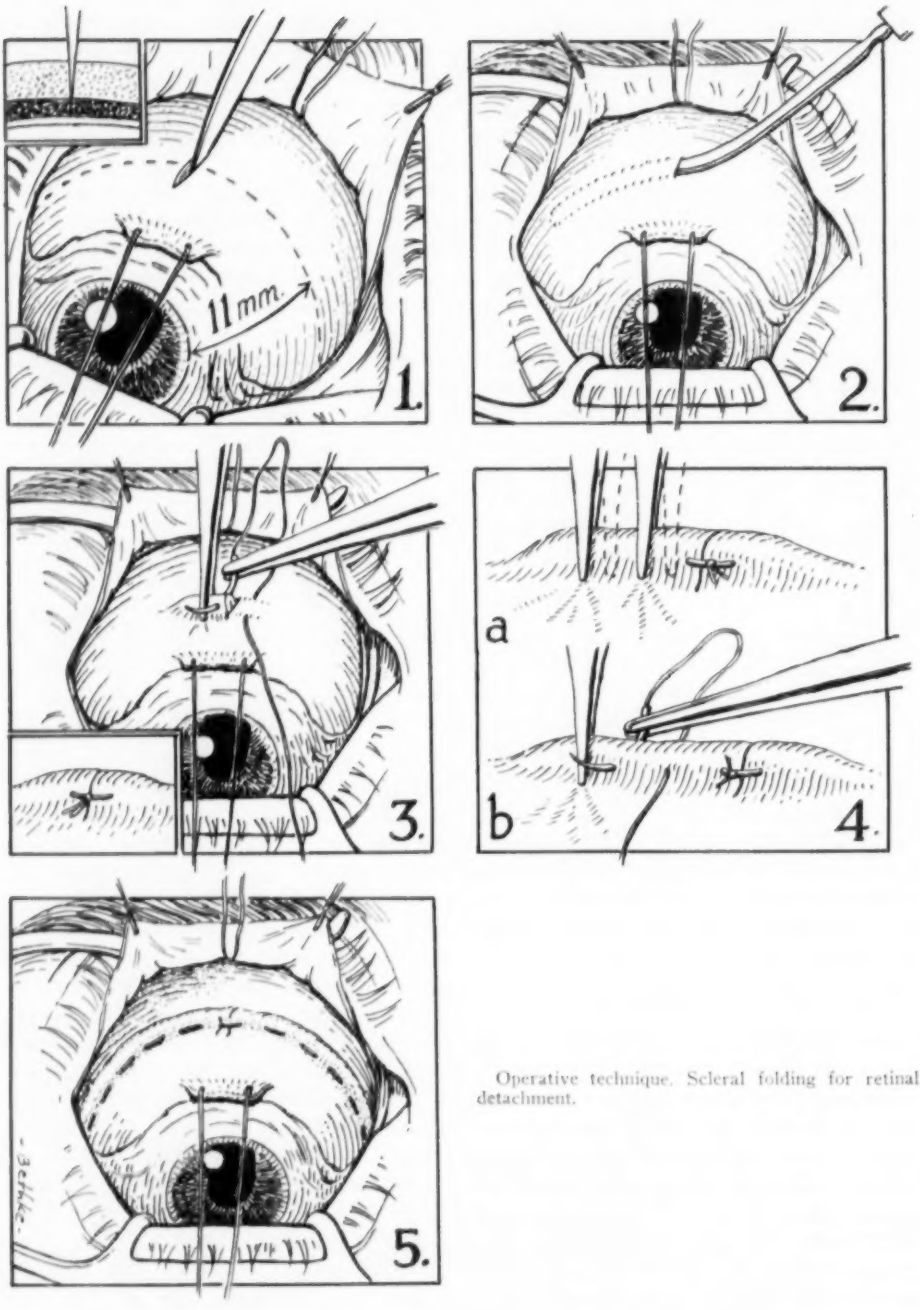
OPERATIVE PROCEDURE IN HUMANS

The operation is performed with the patient under general anesthesia, after routine preparation. The extent and location of the sclera to be shortened are previously determined, as the state of the retina and vitreous indicates. The sclera is adequately exposed through an incision in the conjunctiva and Tenon's capsule, about 10 mm. from the limbus in the desired meridian. It is usually necessary to detach one of the rectus muscles in order to allow folding throughout 180 degrees. A knife incision of the sclera is made down to the choroid (Fig. 1). This incision is about 2.5 mm. long, parallel to the visual axis, and is placed 11 mm. from the limbus, midway between the extremes of the area to be folded.

The globe should be decompressed before the fold is started. This may be achieved by releasing subretinal fluid, either at the scleral wound site or at any area where subretinal fluid exists. A penetrating diathermy electrode and cutting current are utilized for this purpose. After reduction of the intraocular pressure, a spatula is introduced in the scleral incision for separation of the sclera and choroid. This separation is performed in the direction of the planned fold (Fig. 2). Care must be used to hug the sclera with the spatula to prevent choroidal hemorrhage or injury. The first suture is now placed, in a mattress fashion, embracing both lips of the scleral incision in such a manner that, when the suture is tied, an outward fold of the sclera is created, and the scleral wound is tightly closed (Fig. 3).

In completing the fold, particular care must be employed that the choroid is not crimped up with the sclera. This may be avoided by the use of the two Lester type forceps, in the following manner: The end of the fold, which tapers off to normal sclera, is grasped with one forceps, the fold being extended slightly. Then, with the second forceps the sclera is grasped near the first forceps, and the fold is further extended (Fig. 4). This is repeated until the fold has been extended about 2.5 mm.

It is essential that the last bite with the forceps be maintained, thus holding back the choroid, while another suture is placed in a mattress fashion (Fig. 4*b*). A bit more



Operative technique. Scleral folding for retinal detachment.

sclera than is necessary may be taken up in the fold so that the suture can be placed a trifle above the end of the forceps and the choroid not punctured. The fold is continued in this manner to the desired length (Fig. 5).

After the scleral fold is completed,* surrounding diathermy treatment is advisable, as in other shortening procedures, to obtain a mild chorioretinitis. Air may be injected into the vitreous cavity if the eye is unduly soft or if excessive detachment persists.

The detached rectus muscle is reattached, and the conjunctiva is closed.

COMMENT

This operation, when performed on humans, is thought to be safe and technically not difficult. It offers a certain amount of flexibility in that one may alter the extent of the shortening at any time without prior commitment. Such is not the case when a strip of sclera has been excised before closing the wound. Also, if the first suture is securely placed, one should never lose vitreous, for the globe is closed during the remainder of the operation.

The clinical material used in these studies does not lend itself to early appraisal as yet. Observation and study of many cases will be necessary for proper evaluation of this technique.

*Since this paper was submitted, a modification has been added in that the folded sclera distal to the sutures is excised with scissors after the fold has been completed.

SUMMARY

A new type of scleral shortening procedure is presented. Its experimental development and clinical application are discussed. The technical advantages of this operation are mentioned.

Since the preparation of this paper, the author has learned of a similar operation independently developed by Dr. Ramón Castroviejo.

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Ultraviolet Radiation Perimetry with Monochromatic Blue Stimuli

A Method for Early Detection of Conduction Disturbance in the Retina and Optic Nerve

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and
WILLIAM F. HOYT, M.D., San Francisco

The use of ultraviolet radiation as the source of illumination in perimetry is comparatively new. As yet only a preliminary report has been published concerning its general and specific applications.[1] Perimetry with ultraviolet (Blacklight) radiation has introduced luminescent test objects in color whose spectral band is so narrow that for practical purposes they are monochromatic stimuli with a saturation and purity of color surpassing any of the previously used papers. Because the luminescent phosphor of the blue test object selectively absorbs only the wave length from 450μ to 475μ, it emits blue light within a very narrow band. It is possible to narrow this wave-length band still further by the use of filters.

This investigation will report clinical observations when monochromatic blue stimuli are used in the examination of the visual field. Normal visual fields for the blue stimuli were first established, after which a variety of visual field defects were examined to determine the areas of usefulness of the method.

The physiology of retinal light perception will be briefly reviewed as it applies to this investigation, and a theory will be presented to explain the clinicopathological findings.

When luminescent monochromatic blue test objects of relatively large size are used in the examination of the visual field, disturbances of a pathological nature in nerve fiber conduction can be demonstrated in patients with otherwise normal visual fields and with normal visual acuity as tested by Snellen letters. Central scotoma detected with difficulty in the 1/2000 white isopter can be easily demonstrated with these blue stimuli.

Blacklight perimetry is best performed in a dark or semidark room.[1] The retina being tested will therefore be in a different state than when tested under standard illumination. It will be scotopic or partially scotopic rather than photopic. Certain features of the scotopic retina must be considered in order to predict or interpret visual field changes demonstrated under scotopic or partially scotopic conditions.

The scotopic retina is more sensitive to certain wave lengths of light and less sensitive to other wave lengths than the photopic retina. Purkinje * described this phenomenon when he observed that colors at the blue end of the spectrum are most easily perceived at night. When a red and a blue light of equal luminosity in daylight are observed at night, the blue light seems brightest. The visual threshold of blue light is higher in daylight and lower in reduced illumination. The reverse is true of red light. The Purkinje phenomenon explains some of

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* Purkinje,[2] cited by Duke-Elder,[3] pp. 889-899.

the differences between visual fields examined with colored test objects in standard illumination and those examined with ultraviolet (Blacklight) radiation in low illumination.

The luminescent blue test objects used in this investigation emit a virtually pure wave length of light (approximately 470μ), to which the scotopic retina is extremely sensitive.

From an anatomic point of view, it is recognized that the scotopic retina is not equally sensitive in all areas to all wave lengths of light. The fovea is entirely composed of cones, whereas the peripheral retina is almost completely made up of rods. In the scotopic retina the light thresholds for the two types of receptors are different, cones being most responsive to light at the red end of the spectrum and rods to light at the blue end of the spectrum. The relationship of these thresholds is illustrated in Figure 1.

The graph shows the relative sensitivity of the scotopic central and the scotopic

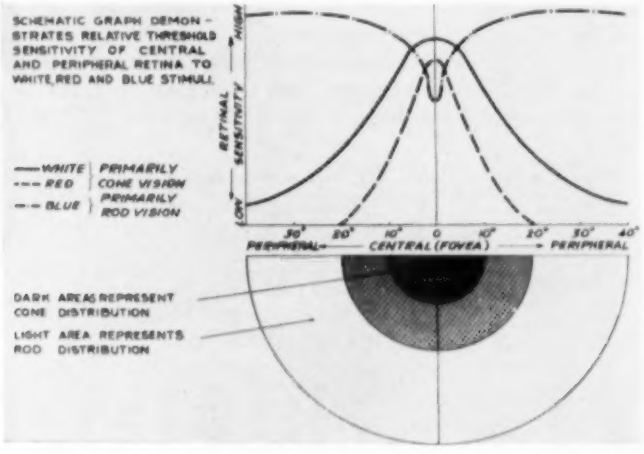
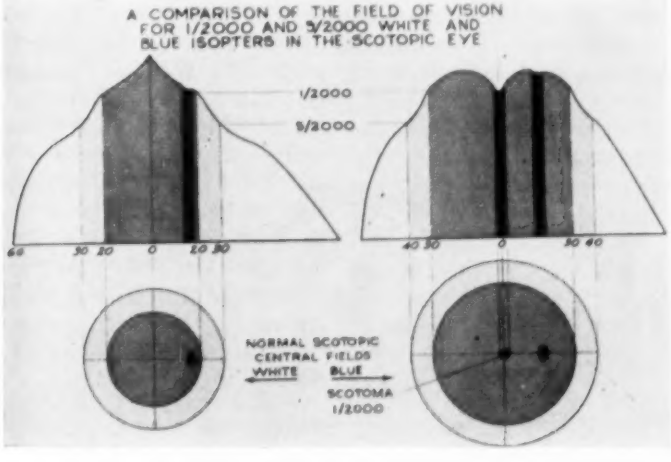


Fig. 1.—Correlation between retinal rod and cone distribution and threshold sensitivity of the scotopic peripheral and central retina to white, red, and blue stimuli.

Fig. 2.—Comparative visual fields for blue and white stimuli in the normal scotopic eye.


A COMPARISON OF THE FIELD OF VISION FOR 1/2000 AND 5/2000 WHITE AND BLUE ISOPTERS IN THE SCOTOPIC EYE
1/2000
5/2000
60 30 20 0 20 30
40 30 0 30 40
NORMAL SCOTOPIC CENTRAL FIELDS
WHITE
BLUE
SCOTOMA 1/2000

peripheral retina to white, red, and blue light. Note that the scotopic peripheral retina is many times more sensitive to blue wave lengths than to red or white (mixed) wave lengths. On the contrary, the scotopic fovea is most sensitive to red and white light (reverse Purkinje phenomenon). Thus, in the scotopic eye the blue and white fields are not comparable, and Figure 2 illustrates the differences.

It will be seen that there is actually a relative central scotoma for light at the blue end of the spectrum,† and this physiologic central scotoma for blue may be clinically demonstrated in the normal eye with very small stimuli. It is also evident that small blue objects have a wider isopter in comparison with white stimuli of equal size.

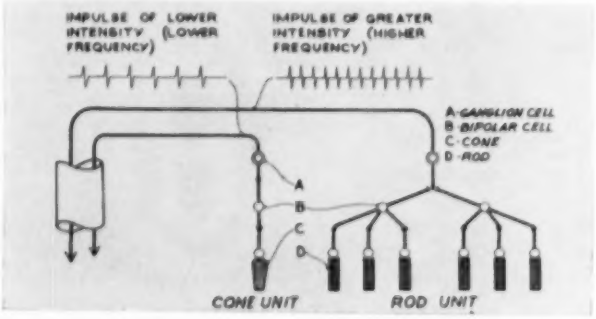


Fig. 3.—The phenomenon of summation. A diagrammatic representation of its effect on impulse intensity and frequency in the retinal and optic nerve fibers.

The explanation for the differential sensitivity of the peripheral and the central retina to light of blue wave length is found in decreasing concentration of rods from the periphery to the fovea. Also, there is a basic anatomical difference in the rod unit and cone unit receptor mechanism.[5] The rod units are made up of a number of receptor cells connected to the same bipolar cells, and a number of these, in turn, are joined to a single ganglion cell. The cone units, in contrast, are composed of a single receptor cell connected to a single bipolar cell, and thence to a single ganglion cell. Rod units, with many receptors transmitting impulses through a common pathway, the ganglion cell, are capable of initiating a much "stronger" (higher-frequency) [5] impulse than is the cone unit, with its single receptor. This is the phenomenon of summation.

The belief that the physiologic relative central scotoma for blue is due to absorption of the blue wave lengths by the yellow pigment of the macula is erroneous. Thompson ‡ demonstrated that the central fovea is free of pigment.

Finally, comment should be made concerning color perception and its bearing upon the method of central field testing under discussion. Much of the difficulty encountered in examination of the color field in photopic eyes arises from the following facts: 1. The colors are not pure. 2. The patient is required to identify the stimulus only when it assumes its true color. The subjective determination of this "end-point," or change from achromatic light and form perception to color determination, is difficult, and it is largely for this reason that color perimetry is considered inaccurate and has been almost abandoned as a clinical method of examination. Color is not a physical property of light. Its perception is a cerebral interpretation. To quote Duke-Elder [8]: "Color is not an attribute of the object, nor the light, nor the eye, but a physical phenomenon which constitutes a quantitative entity only in consciousness."

† References 3 and 4.

‡ References 6 and 7.

Stimulation of the retina with the luminescent monochromatic blue (470μ) test objects used in this investigation must be thought of as an almost pure wave length stimulus. Patients are required not to identify a color but only to state when they see the test object and when it vanishes. Color perception is a cone mechanism.[4] When the intensity of a photic agent impinging upon the rod and cone layer of the retina sinks below the relatively high cone threshold, the perception of color mediated by the cones ceases, leaving only a colorless or achromatic form of light perception.

The method used for visual field examination with luminescent monochromatic blue stimuli and ultraviolet (Blacklight) radiation is simple and readily adapted to the standard equipment in general use. The regulation black-felt tangent screen is used, and the examination is carried out in a dark or semidark room. Patients are tested before dark adaptation is complete, and the examination is usually completed within 5 to 10 minutes, a time insufficient for full dark adaptation. In a few instances more complete dark adaptation was allowed without significant alteration in the results of the examination. As a rule the patient's eyes are rendered only partially scotopic. Only central fields were tested in this investigation. A 5 mm. white luminescent spot was used for fixation and the following isopters were examined: 1/2000, 2/2000, and 5/2000 white and 5/2000 and 10/2000 blue. The test objects were printed on black cardboard disks in luminescent sulfide inks and were illuminated by a 6-watt ultraviolet lamp hand-held by the examiner at approximately 30 in. from the screen.[1]

No color recognition is required in the examination, so that the method is not comparable to color perimetry in the usual sense. The patients are never asked to identify the blue objects as blue but are required only to respond when the object appears or disappears, in the same manner as they respond to the white stimuli. In numerous instances the blue test objects were intermingled with white objects, and there was no verbal suggestion that they might differ in any respect.

CLINICAL OBSERVATIONS, EVALUATION, AND REPORT OF CASES

When luminescent sulfide inks, activated by ultraviolet radiation, were first applied to clinical examination of the visual field,[1] it was felt that the use of the colored inks might, by selective absorption of the light, make possible a color test of the visual field which was more accurate because the colors would be purer than any hitherto available. Such is the case. The narrow spectral band of color emitted by these inks makes them almost truly monochromatic and thereby makes available as a clinical method of examination what has heretofore been purely a research technique.

The first clinical application of monochromatic blue test objects was in an examination of the visual field of a patient who manifested the clinical symptoms of early optic neuritis in the left eye but had a visual acuity of 20/20. No scotoma could be demonstrated in the 1/2000 isopter for white either with standard or with Blacklight illumination, but, because of the complaint of blurring of vision and pain on rotation of the eye, it was felt that a visual field defect should be demonstrable and 5 and 10 mm. luminescent blue objects were used at 2000 mm. A large central scotoma was easily demonstrated in the visual field of the left eye. There were no symptoms in the right eye, and no scotoma could be demonstrated with either 1 mm. white or blue test objects down to 2 mm. in diameter.

Because a physiologic scotoma is known to exist for small blue stimuli, it became necessary to explore the normal visual field for these luminescent monochromatic blue objects.

Twenty normal subjects (ages 22 to 65) with normal visual acuity were tested on the tangent screen at 2000 mm. It was found that, with two exceptions in the older age group, all subjects with 20/20 vision could see a 2 mm. blue stimulus over the entire central field and 25 degrees peripherally. The two exceptions demonstrated a dimming of the 2 mm. blue object at the fixation point. Five-millimeter objects could be seen beyond 30 degrees, and there was no central scotoma. A central scotoma in the younger age group (actual disappearance

of the test object) was demonstrable only with blue objects as small as 1 mm. at 2000 mm. Examination on the perimeter showed the 1/330 isopter for blue to cover the entire visual field. From these observations on normal subjects it was felt that if blue stimuli as large as 5 or 10 mm. were used to test patients with good central visual acuity the normal, or physiologic, scotoma for blue would not be confused with a scotoma from pathologic causes. Visual fields were also examined in normal eyes whose vision had been reduced to 20/100 with plus lenses. The physiologic scotoma for blue was barely demonstrable with 2 mm. stimuli and was not found with 5 mm. blue test objects at 2000 mm.

It was logical to assume from the above that any scotoma for blue which could be demonstrated in the 5/200 or 10/2000 isopters in patients with central visual acuity of 20/20 to 20/30 was definitely pathologic in nature.

Blue test objects were also used in a variety of clinical conditions with manifest visual field defects, and the findings were compared with those with white test objects to determine the conditions in which the monochromatic blue stimuli were most useful. The accompanying Table summarizes this experience.

As might be suspected from the earlier discussion on physiology, the most useful application of the blue stimulus is in disease associated with reduction of central visual acuity. Furthermore, the method is of greatest value when applied to early lesions, and before any defect is detectable with white stimuli.

These central visual field defects are most effectively demonstrated when they are confined to the area within 5 degrees of fixation. The two pathologic disturbances involving this area are macular lesions and lesions of the optic nerve.

Three clinical observations were of special interest in testing for these central visual field defects: 1. Pathologic scotomata for the monochromatic blue stimuli were found before the appearance of any demonstrable scotoma for white in early or minimal lesions of the optic nerve. 2. Some early macular lesions showed much larger scotomata when tested for blue than when even very small white stimuli were used. 3. The subjective ease with which patients uniformly reported the appearance and disappearance of the ultraviolet-radiation-activated blue stimuli was in sharp contrast to the indecision encountered with small white stimuli, especially in standard illumination. Without exception, the subjective responses of patients to the appearance and disappearance of the relatively large blue test objects were quick and accurate on repeated examination, so that the demonstration of even very small central defects was much simplified clinically.

The following case reports, with visual field charts, are illustrative of the superiority of tangent screen scotometry with ultraviolet-radiation-activated monochromatic blue

Comparative Evaluation of White and Monochromatic Blue Perimetry in Lesions of the Retina and Optic Nerve

Site of Lesion	Clinical Findings	Clinical Evaluation
Peripheral retina	Visual field defects detected most readily with white stimuli; no defects demonstrated with blue which could not be shown equally well with white	Usually poor for blue
Central retina	Most central field defects more easily demonstrated with 5 mm. blue than with 1 or 2 mm. white; when found with both white and blue, the defects were the same size but were more readily appreciated with blue stimuli	Excellent for blue
Optic nerve head	Papillitis showed scotoma for blue before it was detectable for white; glaucomatous atrophy showed a scotoma for blue before any defect for white could be demonstrated; papilledema showed the same blind-spot enlargement for blue as for white but no central defect	Excellent for blue
Retrobulbar optic nerve	Early neuritis showed consistent central scotomata for relatively large blue stimuli before any loss of central vision and before detection of any scotoma with white test objects; even when loss for white occurred, it was less striking than the scotoma for blue	Excellent for blue

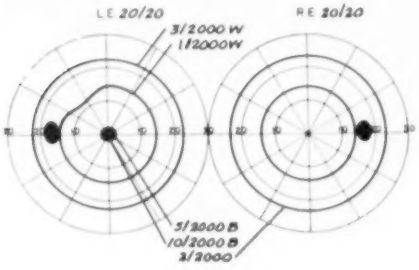


Fig. 4.—Case 1

stimuli in the detection of early and minimal pathologic lesions involving the macula and optic nerve.

REPORT OF CASES

Case 1.—Miss Q.; age 19. Diagnosis: retrobulbar neuritis.

Symptoms.—Pain in left eye on rotation of eye and a vague blurring of left vision, of two days' duration.

Examination.—Vision 20/20, in each eye, but some dimness of letters with left eye. Ophthalmoscopic examination negative.

Visual fields showed no defect in 1/330 and 1/2000 white isopters with both standard and ultraviolet illumination. Isopters for 5/2000 and 10/2000 monochromatic blue stimuli showed dense and easily demonstrated central scotoma in left visual field (Fig. 4). No scotoma was demonstrated with blue test object in standard illumination. Right visual field showed no defect for any test object, either white or blue, with standard or ultraviolet illumination. Reexamination in two weeks showed no symptoms, and left central scotoma for monochromatic blue stimulus had disappeared.

Case 2.—Mrs. P.; age 32. Diagnosis: retrobulbar neuritis (multiple sclerosis).

Symptoms.—Severe pain on ocular rotation, left eye, of three days' duration. Euphoria and reflex changes indicative of multiple sclerosis.

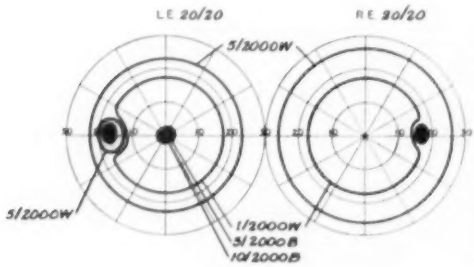


Fig. 5.—Case 2

Examination.—Corrected vision 20/20, each eye. Ophthalmoscopic examination normal. Visual fields showed no defect for 1/2000 white isopter either with standard or with ultraviolet illumination (Fig. 5). Isopters for 5/2000 and 10/2000 monochromatic blue stimuli showed central scotoma and slightly enlarged blind spot in left eye. The right visual field was normal.

Reexamination two weeks later showed a small but demonstrable central scotoma for 1/2000 white with gross enlargement of the defect for blue and more advanced signs of multiple sclerosis.

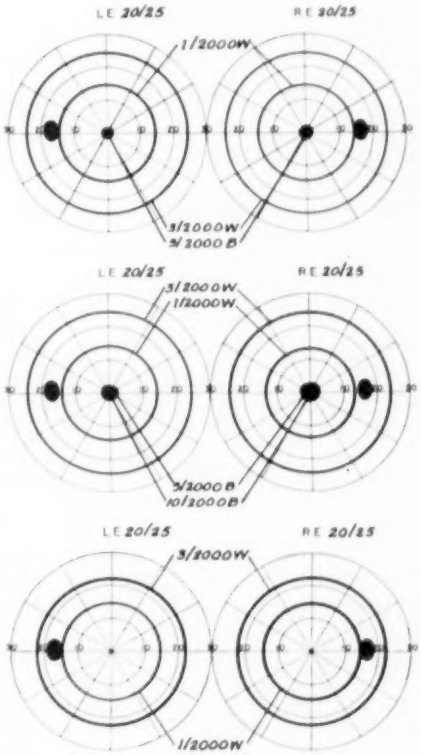


Fig. 6.—Case 3

Case 3.—Mr. K.; age 42. Diagnosis: probable transient retrobulbar neuritis.

Symptoms.—Diplopia and vague blurring of vision. Severe exophthalmos. Patient undergoing treatment with daily x-radiation directed through the skull to the pituitary from both sides.

Examination.—Visual acuity 20/25, each eye. Ophthalmoscopic examination normal in each eye. Visual fields normal in each eye for 1/2000 white isopter with both standard and black light (Fig. 6). Definite bilateral central scotoma for 5/2000 monochromatic blue isopter. One week later, after

further pituitary radiation, the scotoma for blue was even larger, though it was still not possible to demonstrate a defect for white. One month after discontinuance of x-ray treatment the exophthalmos had regressed considerably; the patient was symptomatically improved, and the central scotomata for the monochromatic blue stimuli had disappeared.

Case 4.—Mr. B.; age 54. Diagnosis: retrobulbar neuritis.

Symptoms.—Chronic ethmoid and frontal sinusitis with fistulous tract (demonstrated by x-ray) into posterior portion of right orbit.

Examination.—Visual acuity 20/20, each eye. Moderate proptosis of right globe. Ophthalmoscopic examination normal. Visual fields demonstrated no defect in the 1/2000 white isopter for standard or black light, but a definite scotoma in the right field was found in the 5/2000 isopter for monochromatic blue (Fig. 7). At operation an orbital abscess was found extending throughout

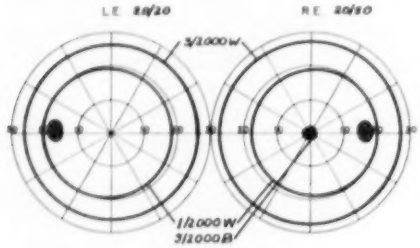


Fig. 7.—Case 4

the orbit and surrounding the right optic nerve. Three weeks after operation the proptosis had subsided and the central scotoma for blue was no longer demonstrable.

Case 5.—Mrs. S.; age 29. Diagnosis: optic neuritis secondary to cellulitis of face.

Symptoms.—Severe facial cellulitis secondary to osteomyelitis of the maxilla. Pain on ocular rotation and slight blurring of right vision.

Examination.—Visual acuity: right eye 20/25; left eye 20/20. Ophthalmoscopic examination normal. Edema of face and eyelids on right side. Visual fields showed a minute, indefinite, and inconstant central scotoma in the 1/2000 isopter for white in the right eye. With the monochromatic blue stimuli there was a large and easily outlined central scotoma in the 5/2000 and 10/2000 isopters (Fig. 8).

Case 6.—Mrs. T.; age 35. Diagnosis: retrobulbar neuritis; multiple sclerosis.

Symptoms.—Well-advanced multiple sclerosis with motor and sensory involvement.

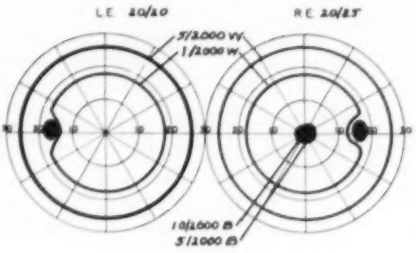


Fig. 8.—Case 5

Examination.—Visual acuity: right eye 20/70; left eye 20/200. Ophthalmoscopic examination showed bilateral optic atrophy of moderate degree. Visual fields showed bilateral central scotomata in the 1/2000 and 2/2000 white isopters, with a somewhat larger scotoma demonstrated for 5/2000 blue (Fig. 9).

Case 7.—Mr. V.; age 53. Diagnosis: multiple sclerosis.

Symptoms.—Haziness of vision in the left eye, of several months' duration. Periodic ataxia and sensory disturbances in extremities.

Examination.—Visual acuity: right eye 20/20; left eye 20/30. Ophthalmoscopic examination revealed a slight pallor of the left optic disc. Visual fields showed a normal field in the right eye for all isopters. The left field was generally depressed in the 1/2000 white isopter but no scotoma was found. There was a definite and easily demonstrated scotoma in the left field in the 5/2000 isopter for monochromatic blue (Fig. 10).

Case 8.—Mrs. B.; age 40. Diagnosis: optic atrophy secondary to neuritis.

Symptoms.—Visual loss of long standing in the right eye.

Examination.—Visual acuity: right eye, hand movements; left eye, 20/20. Ophthalmoscopic examination showed marked pallor of the right optic disc with blurred margins. Visual fields (Fig. 11) showed a large, dense central scotoma in the right

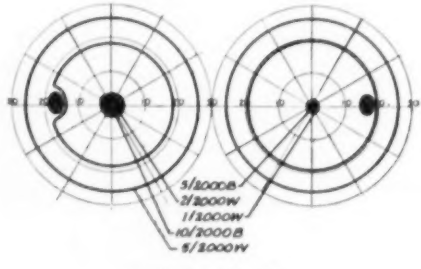


Fig. 9.—Case 6

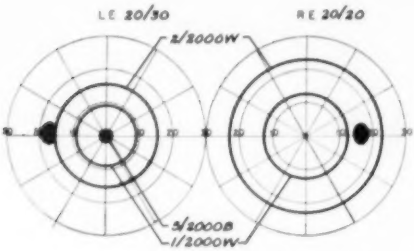


Fig. 10.—Case 7

field for all test objects, of the same size for blue and white. The left field was normal.

Case 9.—Mrs. W.; age 63. Diagnosis: bilateral senile macular degeneration.

Symptoms.—Gradual decrease in reading vision.

Examination.—Visual acuity: right eye 20/50; left eye 20/20. Ophthalmoscopic examination showed senile macular degeneration with some macular edema in the right eye and minor pigmentary changes in the left eye. Visual fields

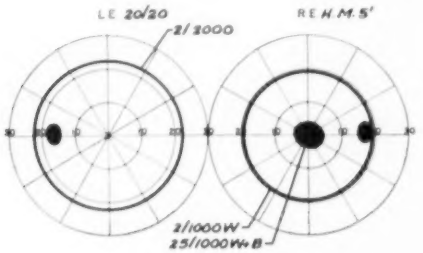


Fig. 11.—Case 8

(Fig. 12) showed a vague and indefinite central scotoma in the right field for the 1/2000 white isopter and a small but definite central scotoma for the 5/2000 blue isopter in the same eye. The left field was normal for all test objects.

Case 10.—Mrs. M.; age 33. Diagnosis: central angiospastic retinopathy.

Symptoms.—Blurred vision and severe metamorphopsia.

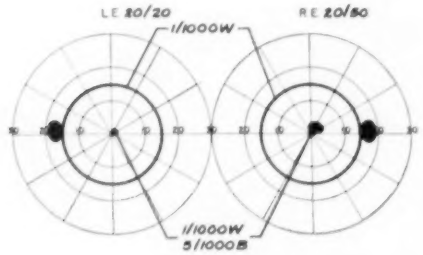


Fig. 12.—Case 9

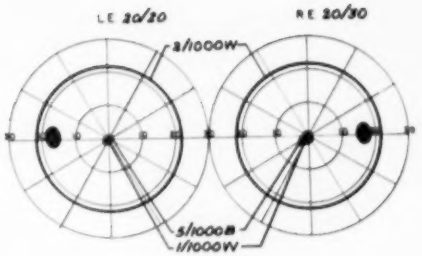


Fig. 13.—Case 10

Examination.—Visual acuity: right eye 20/30; left eye 20/20. Ophthalmoscopic examination showed a pigmentary disturbance in each macula, with some edema. Visual fields (Fig. 13) showed a minute central scotoma, demonstrated with much difficulty, in both eyes with the 1/2000 white isopter. The 5/200 monochromatic blue isopter showed a definite, sharp-bordered central scotoma.

Case 11.—Mr. P.; age 71. Diagnosis: bilateral macular hole.

Symptoms.—Blurring of central vision, which the patient attributed to exposure to a welding flash 28 years before.

Examination.—Visual acuity: right eye 20/30; left eye 20/50. Ophthalmoscopic examination showed bilateral small round macular holes. Visual fields (Fig. 14) showed very small and indefinite central scotomata in the 1/2000 and 2/2000 white isopters. The same scotomata, very slightly larger, were demonstrated with ease in both the 5/2000 and the 10/2000 blue isopters.

Case 12.—Mrs. G.; age 78. Diagnosis: open-angle glaucoma with glaucomatous optic atrophy.

Symptoms.—Gradually decreasing vision, of many years' duration.

Examination.—Visual acuity: right eye 20/20; left eye 20/200. Visual fields (Fig. 15) show generalized depression and typical nerve fiber bundle defect in the left eye for fairly large white test objects, but the right field was normal in the 1/2000 white isopter. The right field, however,

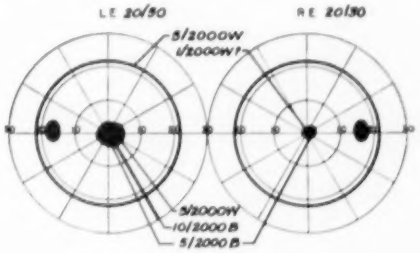


Fig. 14.—Case 11

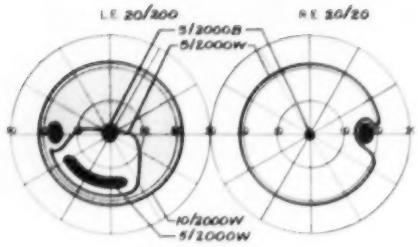


Fig. 15.—Case 12

shows a definite and easily demonstrated central scotoma in the 5/2000 isopter for monochromatic blue.

Case 13.—Mr. S.; age 65. Diagnosis: open-angle glaucoma.

Symptoms.—Decreasing vision in left eye.

Examination.—Typical open-angle glaucoma in both eyes, with elevated intraocular pressure controlled by miotics. Both optic nerve heads deeply cupped and atrophic. Visual acuity: right eye 20/20; left eye 15/200. Visual fields (Fig. 16) showed typical glaucomatous defects in the left eye, but inconclusive visual field changes in the right eye when tested with white stimuli. The right field showed a marked central scotoma for the 5 mm. monochromatic blue test object.

Case 14.—Mr. R.; age 72. Diagnosis: open-angle glaucoma.

Symptoms.—Slight blurring of vision.

Examination.—Visual acuity: right eye 20/30; left eye 20/25. Ophthalmoscopic examination showed marked cupping and some atrophy of both optic discs. Visual fields (Fig. 17) showed definite glaucomatous defects for white stimuli in the right eye but none in the left eye. Both fields showed central scotomata in the 5/2000 and 10/2000 isopters for monochromatic blue.

Case 15.—Mrs. J.; age 56. Diagnosis: open-angle glaucoma.

Symptoms.—None.

Examination.—Visual acuity 20/25, each eye. Intraocular pressure varied between 32 and 40 mm.

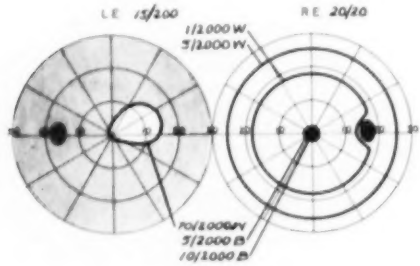


Fig. 16.—Case 13

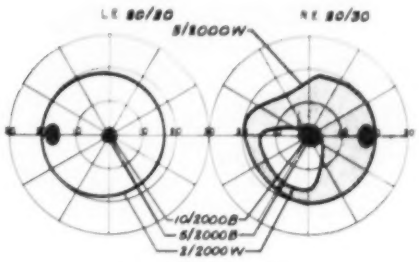


Fig. 17.—Case 14

(Schiøtz) and was not controlled with miotics. Ophthalmoscopic examination was normal. Visual fields (Fig. 18) showed no defects in the 1/330 and 1/2000 isopters for white, but both fields showed a consistent central scotoma for 5/2000 and 10/2000 monochromatic blue. After several months a nerve fiber bundle defect developed in the left eye, which could be demonstrated with the small white stimuli. At the same time the scotoma for blue increased in the left eye.

Case 16.—Mrs. C.; age 65. Diagnosis: open-angle glaucoma.

Symptoms.—Haziness of central vision.

Examination.—Visual acuity 20/25, each eye. Intraocular pressure 37 mm. (Schiøtz), reduced to 25 mm. (Schiøtz) with pilocarpine. Bilateral very early incipient cataract. Ophthalmoscopic examination showed bilateral glaucomatous cupping of the optic discs, with optic atrophy. Visual fields (Fig. 19) showed a dense arcuate scotoma in the left eye with white stimuli and large and easily

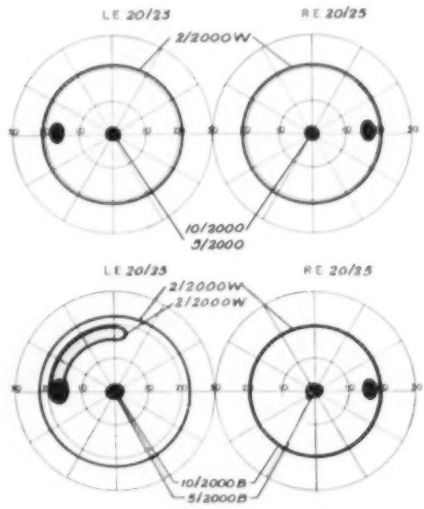


Fig. 18.—Case 15

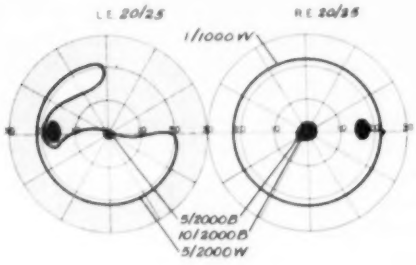


Fig. 19.—Case 16

demonstrated bilateral central scotomata in the 5/1000 and 10/1000 isopters for monochromatic blue.

CASE 17.—Mrs. C.; age 55. Diagnosis: open-angle glaucoma.

Symptoms.—Blurring of vision, right eye; absence of left eye.

Examination.—The left eye had been enucleated. The right eye had had an iridencleisis, with control of increased intraocular pressure. Visual acuity was 20/30. Visual field (Fig. 20) showed general depression for the 2/1000, 2/1500, and 2/2000 white isopters but no characteristic glaucomatous defect. There was a definite central scotoma for 5/1000 monochromatic blue.

CASE 18.—Mr. F.; age 40. Diagnosis: commotio retinae, left eye.

Symptoms.—Defective vision in left eye since gunshot wound in left temple and orbit three months previously. There was immediate loss of all vision, followed by gradual improvement.

Examination.—Visual acuity: right eye 20/20; left eye 20/200. Ophthalmoscopic examination was normal in the right eye. The left eye showed a round, sharply outlined hole in the macula, surrounded by an area of retinal edema. The temporal peripheral retina showed a large area of pigmentation with some residual retinal hemorrhages and exudate. The visual field (Fig. 21) in the left eye showed a small, sharply outlined central scotoma for 1 mm. white test objects with both standard and ultraviolet illumination. Within the 10/1000 isopter for monochromatic blue was a large, dense, and much more easily demonstrated central scotoma.

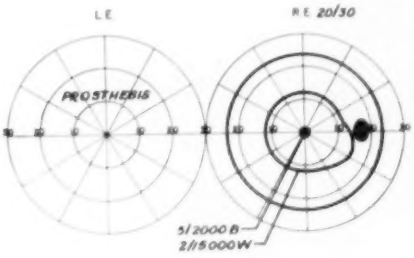


Fig. 20.—Case 17

COMMENT

Any technique of perimetry which simplifies the demonstration of a visual field defect from the patient's point of view is of clinical value. Subjective response to the disappearance of these luminescent, monochromatic blue test objects was quick and accurate, and confusion was minimal. Because of the higher sensitivity of the paracentral and peripheral retina to blue light, perception of the test object was easy in the peripheral field, especially when contrasted with small white stimuli. There was no need to concentrate subjectively in order to see these large blue stimuli, whose disappearance into scotomatous defects was sudden, dramatic, and easily reported.

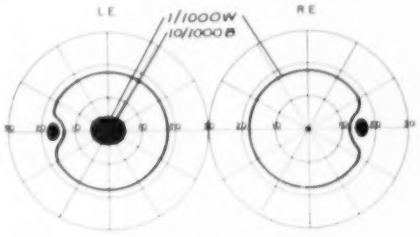


Fig. 21.—Case 18

Beyond being a simple and generally better way of demonstrating central visual field defects, the use of monochromatic blue stimuli activated by ultraviolet radiation appears to have value in demonstrating early pathologic changes in the macula and in the optic nerve before these changes or their associated field defects can be demonstrated with white test objects.

From analysis of the cases studied and the visual field studies on normal eyes, it appears quite certain that these scotomata found within the 5/2000 and 10/2000 isopters for monochromatic blue are pathologic, and not physiologic, defects.

Confusion with the normal or physiologic scotoma for blue is avoided merely by using test objects sufficiently large not to be ob-

scured in the central fields of normal persons. As a further precaution, the method was used only with patients whose visual acuity by Snellen letters was 20/30 or better. One source of error is known to be present in patients with senile lenticular changes, when a scotoma for blue may develop as the result of absorption of the blue lengths by cataractous lenses. The error is obvious and easily avoided.

With the discovery of a pathologic central scotoma, it is desirable to localize the lesion which produced it, either in the receptor elements at the macula or in the conducting fibers of the nerve fiber layer of the retina or optic nerve. Aside from the usual differentiating signs and symptoms, such as associated pain on ocular rotation in optic neuritis or metamorphopsia in macular disease, the form of the scotoma itself helps to localize the lesion. It was found that the scotomata for blue which were due to macular disease were typically irregular, eccentric, or paracentral, whereas the scotomata produced by lesions of the optic nerve or edema of the retinal nerve fiber layer were symmetrical, round, and centrally placed.

It is interesting to consider the etiology of these pathologic scotomata for blue light in the scotopic eye. Clinically, it would appear that visual field defects for blue stimuli in the absence of like defects for white are evidence of an early disturbance in the retinal receptor or conduction mechanism. These two sites have one element in common: the nerve fiber or axon of the ganglion cell. Amsler,[9] in his discussion of early macular changes, mentions that a translucent scotoma is sometimes demonstrable and states that it is caused by a fluid collection in the superficial layers of the retina in the macular area. This fluid accumulation may disrupt or impede transmission of impulses along the axons of the ganglion cells. For the same reason, early changes in the optic nerve may present a similar picture.

If it is assumed that these visual field defects for blue stimuli are due to nerve fiber lesions, then why do they not present nerve fiber bundle defects of the type with which we are familiar? On purely physiologic grounds it can be argued that the impedance or blocking of stimuli in the nerve fibers could be generalized to the pathologic process but the effect of interference would be different in fibers carrying impulses from rod areas from that in fibers carrying impulses from the cone areas. The rod areas, because of the phenomenon of summation, are able to send stronger stimuli of higher frequency through their nerve fiber axons. The general slowing or impedance to transmission of stimuli along the nerve fibers in the optic nerve in early disease may be just enough to screen out the weaker or lower frequency impulses from the cone areas, and a scotoma appears. Blue stimuli viewed centrally in the scotopic eye are very close to the threshold of perception and therefore send weak or low frequency impulses through the nerve. Central scotomata for monochromatic blue stimuli may therefore represent very early

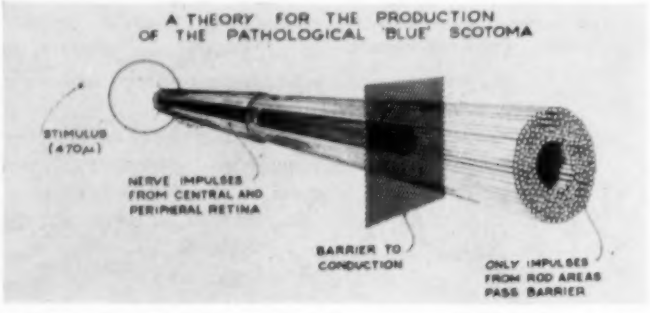


Fig. 22.—Schematic representation of the effect of a conduction barrier in screening out low-frequency (cone) impulses in the optic nerve to produce a pathologic central scotoma for monochromatic blue stimuli.

evidence of conduction interference in the nerve fibers.

The theory is supported by the clinical findings presented in this paper. The use of monochromatic blue stimuli, consisting of luminescent ink activated by ultraviolet radiation to emit a light of approximately 470μ wave length constitutes a clinical technique in perimetry which is of great value in the diagnosis of early lesions of the nerve fibers of the retina and optic nerve with conduction interference in these fibers.

SUMMARY

The use of ultraviolet (Blacklight) radiation and luminescent test objects in perimetry has reopened the field of color perimetry as a practical, and more objective, clinical method of examination.

The application of colored luminescent inks to test objects has made it possible to produce color stimuli which are virtually monochromatic and of greater purity and saturation than any of the papers previously used. With these inks, activated by ultraviolet radiation, a very narrow wave-length band is utilized as a stimulus in the visual field examination. In the case of the blue color the wave-length stimulus falls within the band from 450μ to 475μ.

The physiology of light and color perception and the visual thresholds for color in the photopic and scotopic eye are discussed as they apply to this perimetric technique.

The normal visual field for monochromatic blue stimuli has been studied.

A series of cases is presented which demonstrates the value of the monochromatic blue stimulus, particularly in the early diagnosis of lesions of the nerve fiber conduction mechanism in the retina and optic nerve.

The significance of central scotoma demonstrated with monochromatic blue stimuli in the absence of visual field defects for white is discussed, and a theory for their production is advanced.

384 Post St.

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Ankyloblepharon Filiforme Adnatum

MORTIMER A. LASKY, M.D., Brooklyn

Ankyloblepharon filiforme adnatum, a rare congenital anomaly, and difficult to explain, was first reported by Hasner in 1881. He has the distinction of having named the puzzling developmental defect. Eleven subsequent cases have been added to the literature, with but one appearing in the English language, completely described by Judge, Mott, and Gabriels.[1] Since their article appeared, Mattsson[2] and Lobstein and Haarscher[3] have contributed further reports.

The bands are single or multiple, usually occur monocularly, and are somewhat elastic, although definitely limited in their extensibility. There is no definite relationship to prematurity, as only 1 case of 13 occurred in a 7-month premature infant. Sex has no bearing on the incidence. Most of the bands are located in the lateral halves of the lids; a few are nasally placed. One infant had an associated harelip, and another had a cardiac defect.

The bands are always located between the cilia and the orifices of the Meibomian glands. Histological studies have shown that the structure is essentially a central vascular connective tissue strand surrounded by pavement epithelium. Sectioning of the bands usually results in some brisk bleeding, which is self-controlled, and shrinkage of the stubs, with rapid, complete disappearance of all vestiges of the excess tissues.

The mechanism of production of the adherent bands is still a moot question. Study of the embryonic development of the lids shows that they begin to form at the 16 mm. stage, in the second fetal month, and that they meet, becoming adherent with epithelium at 31 mm., the adherence being firm at 35 mm. Separation begins in the fifth month of gestation, becoming complete by the sixth month.

It has been postulated that an inflammation of the lid margins causes defects in the epithelium, permitting mesenchyme to grow across and thus form firm strands that prevent the normal separation at the sixth fetal month. If this is so, how can one account for the invariable normal state of the cilia and the Meibomian glands, the bands always extending between the gray lines?

Another theory has been that the fingernails of the fetus scratch away epithelium and thus permit mesenchymal proliferation across the line of junction. It is difficult to accept such selective scratching, especially when one considers the fact that the nails are not quite long enough until the last few months of pregnancy, after lid separation normally has occurred.

Judge and associates[1] believed that the uniformity of the bands and their constant origin at the white line indicate "a congenital deviation," and that the anomaly should be classed with other abnormalities of development, such as various types of coloboma, etc.

The following case report is offered as an addition to the literature and is believed to be the second to appear in the English language journals:

Baby B, a white boy, was seen in the nursery of the Jewish Hospital of Brooklyn at 4 days of age. He was born at full term, weighed 2320 gm. (5 lb. 2 oz.), and was delivered after a normal labor with low forceps. He was normal in all respects except for an adherent condition of the lids of the right eye that prevented full separation of the lids (Fig. 1). Examination revealed three thin strands of pale-pink tissue, two at the outer canthus and a broader central band. These ran from gray line to gray line, and the cilia were free of any involve-

From the Ophthalmological Service, Jewish Hospital of Brooklyn.

Fig. 1.—Right eye before surgical intervention.

Fig. 2.—Right eye after surgical intervention.

ment. The underlying bulb was entirely normal, as were the left eyeball and lids. The bands were severed by a Stevens scissors, accompanied by moderate bleeding, which soon ceased. The stubby remnants rapidly shrank, and examination one week later disclosed no marginal pathology (Fig. 2). Observation of the lids one month subsequent to treatment showed normal lids.

SUMMARY

A case of ankyloblepharon filiforme adnatum is reported as the 13th in the world literature and the 2d in the English literature. The author agrees with Judge and colleagues that the condition is a congenital abnormality due to an anomalous development.

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Successful Corneal Transplants in Four Eyes of Two Patients with Lattice Dystrophy

JOSEPH LAVAL, M.D., New York
and
RALPH I. LLOYD, M.D., Brooklyn

The hereditary corneal dystrophies were described in the pre-slit-lamp days by Groenouw (1890), Biber (1890), and Haab and Dimmer (1899). The diagnostic features of granular and macular dystrophy were not evident with the apparatus then available, but the details of lattice dystrophy could be made out with oblique illumination and the loupe and this entity still retains its original name. Under the title of Groenouw's nodular dystrophy, the granular and macular types have been commingled in case reports, but most of these cases were certainly of granular dystrophies, since Bücklers, in 1938, found 96 cases of granular dystrophy, 27 cases of macular dystrophy, and only 6 cases of lattice dystrophy when he made his survey of Württemberg as a part of the plan to eliminate hereditary ailments from the German race.

Lattice dystrophy is a dominant hereditary affliction, first making its appearance about the 10th year of life. Despite the great number of crisscrossing "tubes," the vision is not seriously affected until ulceration begins. Very few microscopic sections have been made, but the sections by Lowenstein and one from one of our patients at the Manhattan Eye, Ear and Throat Hospital confirm the impression gained by clinical observation. The ulcer seems to be the result of an elevated nodule from which the epithelium is rubbed by the lid movements. The vitality of the tissues involved is undoubtedly impaired by a blister-like formation, with destruction of Bowman's membrane and, finally, deeper effects in the stroma. The corneal tissue becomes denser and denser as the attacks of inflammation and ulceration continue.

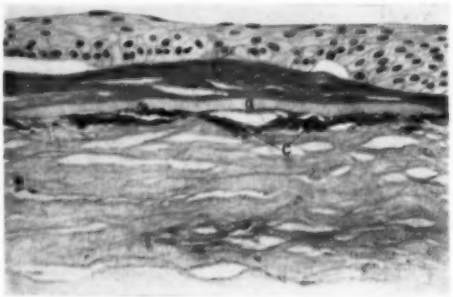

Fig. 1.—Lattice dystrophy (Löwenstein, A.: Klin. Monatsbl. Augenh. **82**:752, 1929; published by Ferdinand Enke, Stuttgard). (*a*) Bowman's membrane; (*b*) acidophile deposit; (*c*) basophile deposit in stroma.

The pathological picture in this type of dystrophy is well shown in the illustration taken from Lowenstein's paper (Fig. 1) and also in the slide prepared from the cornea of Case 1 in the present report (Fig. 2). The hyaline deposit (acidophile deposit of Lowenstein) beneath the epithelium is well defined, and the changes in the epithelial layer are well shown.

Bücklers has shown that the three corneal dystrophies are not interchangeable, nor do various types occur in a family. The granular dystrophy is dominant and affects only the central corneal area, leaving a clear zone

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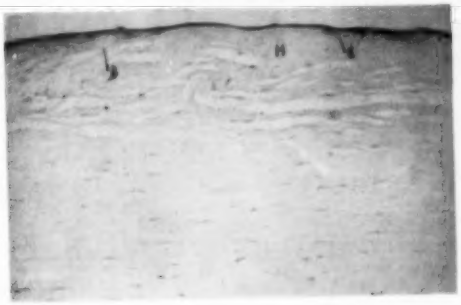


Fig. 2.—Corneal tissue from Case 1. *B*, Bowman's membrane; *E*, epithelial changes; *H*, hyaline deposit.

Fig. 4.—Right eye, 1953, after corneal graft (postoperative).

peripherally and between the various spots and dots. There is usually no ulcer and no inflammation. The lesions are seen as early as 5 years of age, and the vision does not get too poor to allow the patient to do ordinary work. Like the other corneal dystrophies, this form is avascular and bilateral.

The macular dystrophy is recessive, and its effects are most severe, the entire cornea being involved, and even the intervals between the large spots are thickly dotted. Loss of vision is the first symptom that makes the patient know that there is something wrong, in both granular and macular dystrophy. Ulcers occur in the lattice type and are rare in the macular type, but attacks of inflammation do occur. Towne improved the vision in a case of the macular type by contact lenses and made it possible for an affiliated officer to finish his service during the World War on a small boat in all kinds of weather. The efforts made to relieve these patients have included local applications, then scraping and curetting, next superficial transplants, and, at present, lamellar and penetrating grafts.

The number of cases of lattice dystrophy treated surgically is small indeed because of the few cases of the generally accepted idea that the new tissue would soon lose its clarity. Because of the success obtained by one of us (J. L.) in four eyes of two patients, seen first by the other (R. I. L.) in 1933 and photographed in 1938, this presentation is made.

REPORT OF CASES

In February, 1933, Mr. W. P., aged 31, was first seen by R. I. L., complaining of a large superficial corneal ulcer of the left eye. Both corneas were insensitive, and the slit lamp showed many optically empty pipes in various layers of the stroma, too numerous to depict. The ulcers healed readily when ordinary measures were used, plus closing of one or both eyes as the occasion might require. Vision after the first bout was 20/33 and 20/40, with correction. Contact lenses were fitted in 1942, but the

Fig. 3.—Corneal appearance O. D., 1942. Preoperative.

Fig. 5.—Corneal appearance O. S., 1942. Preoperative.

Fig. 6.—Appearance O. S., 1953, after corneal graft (postoperative).

patient would not wear them, although they did improve his vision somewhat. Vision of the right eye was 10/50 with and without the contact lens, but vision in the left eye improved from 10/200 to 10/30. The patient had repeated attacks of ulceration of first one cornea and then the other (Fig. 3). No diagnosis other than corneal ulcer was made until the article by Bucklers appeared, in 1938, at which time the diagnosis of lattice dystrophy was made. In September, 1948, when he gave a history of having had "ulcerated eyes" for the past 16 years, he was seen by J. L. Vision was 20/200 O. D., not improved, and 20/50— O. S. with a contact lens or with +4.00 sph. ⁻ +2.50 cyl, ax 135. Both corneas had a lattice type of dystrophic keratitis, and the right cornea had a central raised, whitish area with serrated edges. This area stained with fluorescein and measured 2×2 mm. It was fairly superficial and seemed to reach just below Bowman's membrane. It was easily removed by a superficial keratectomy. When the tissue was fixed and stained, it was seen to consist of corneal epithelium overlying an amorphous, evenly staining mass. Following this, vision O. D. was improved from 20/200 to 20/50— with +1.00 sph. ⁻ +1.00 cyl, ax 135.

On Dec. 16, 1950, there was recurrence of inflammation in the right eye, and homatropine 1% was

Fig. 7.—O. D., postoperative, October, 1954.

Fig. 8.—O. S., postoperative, October, 1954.

ordered to be used three times daily. Two days later he returned complaining of headache and nausea; the vision O. D. was 20/400, and the intraocular tension was 40 mm. Hg (Schiøtz). After pilocarpine and physostigmine instillation, the tension rose to 60 mm. Hg, and an iris inclusion was performed that same day on the right eye. Thereafter the tension in each eye remained at 20 mm. Hg.

A few weeks later the patient developed a typical epidemic keratoconjunctivitis in the left eye, with a fairly large preauricular gland on the left side. Four weeks later the left eye had quieted, with no evidence of corneal scarring added to the old keratitis, which then had some deep corneal vessels, growing in at the limbus below. Vision in the O. D. (the eye with the iris inclusion) was 20/70 with +1.00 sph. ⁻ +1.00 cyl, ax 120 and O. S. 20/200 with +4.00 sph. ⁻ +3.00 cyl, ax 135 (Figs. 4 and 5).

Four months later, on June 1, a penetrating 5 mm. corneal graft was performed on the left eye, using two Paton overlying sutures. Three weeks later some bulging of the graft above was noted and some grayness of the graft nasally. Later, a few epithelial blebs developed over this area of grayness. The intraocular tension was normal. Beta-ray therapy was given to the grayish area

Fig. 9.—Corneal appearance O. D. (left), 1940, and O. S. (right), 1953, after reoperation.

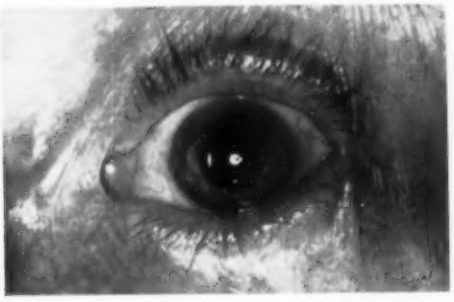


Fig. 10.—O. D., 1954, postoperative.

by Dr. Okrainetz daily for two weeks; one month later the blebs were gone, and the grayness was less apparent. At this time the vision was 20/50 with +1.00 sph. ⁻ +2.00 cyl. ax 120 for the left eye. Cortisone (Corton) drops had been used intermittently and were continued. The tension at one time rose to 30 mm. Hg but was quickly controlled with pilocarpine, after which the tension remained 20-25 mm. Hg without medication. On Aug. 15, two months after the corneal graft operation, vision O. S. was 20/40— with correction, improved from 20/200 preoperatively (Fig. 6).

One year later the tension of the right eye was 25 mm. Hg and the vision was 20/80. On Oct. 24, 1952, a penetrating 5 mm. corneal graft was done on the right eye, using two Paton overlying sutures again. The tension did not rise postoperatively even with atropine. With a —7.00 lens, vision of 20/40 was obtained for the right eye in February, 1953, improved from 20/80 preoperatively. In June, 1954, the vision was O. D. 20/40 with a —7.00 lens and O. S. 20/40+ with +0.50 sph. ⁻ +0.50, ax 90. The patient now uses his left eye for distance vision to drive his car and reads with the right eye without glasses (Figs. 7 and 8).

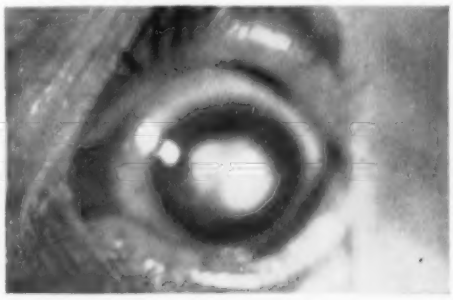
It is remarkable that there have been no more attacks of redness, irritation, and photophobia in either eye since the corneal grafts were performed. Apparently, the new corneal tissue has had a beneficial effect on the underlying pathological process.

Case 2.—Mrs. R. P., white, age 59, mother of Mr. W. P., was seen in 1938 by R. I. L., at which time the other members of the family were also examined. The mother and two sisters were found to have typical corneal lattice dystrophy, and one brother was normal. The mother's vision was 4/200 and 10/200, and the corneas were very white and insensitive. The ulcers appeared first at 30 years of age, but she had not had any exacerbations for the past six years. The slit lamp showed a dense cornea with irregular cotton-white streaks in the stroma, some of which contained blood, a picture quite different from the earlier stages of all these dystrophies, which are avascular. There was nothing to be done about this pitiable case at that time, but after the successful operation on the son, the mother, then in her 70's, demanded the same treatment. She was seen by J. L. on June 18, 1951, at the age of 72. Vision was light perception O. U. with good projection. The tension was 28 mm. Hg O. U. Both corneas were yellowish and opaque, but an anterior chamber could be seen because the opacity did not quite reach to the limbus in either eye (Fig. 9).

On Aug. 2, 1951, a 5 mm. penetrating corneal graft was performed on the right eye. There were no complications, and vision was 20/40 with —9.00 D. six weeks later (Fig. 10). On May 2, 1953, one and a half years later, a similar operation was performed on the left eye. The vision on Feb. 4, 1954, was 20/40 O. S. with —1.50 D. (Fig. 11).

Almost four years have passed since the first eye of these two patients was operated on, but to date all four of the corneal grafts have remained clear. These cases will be kept under observation, and we hope to be able to report further in detail. Reports of only a few corneal transplants in cases of lattice dystrophy are available, and the relief given these patients justifies the surgical procedure.

Fig. 11.—O. S., 1954, postoperative.



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Clinical Notes, New Instruments and Techniques

NEW GERMICIDAL TONOMETER STAND

ROBERT S. ROSNER, M.D., Cleveland

EVERY ophthalmologist is interested in some sanitary method of using the tonometer so that the instrument will not transfer an infection from one patient to the next.

Various techniques have been used. These included wiping the plunger with ether-alcohol mixtures, ether alone, or alcohol alone. The tonometer stand that kept the plunger constantly immersed was discarded because fluid got between the plunger and the footplate and rose by capillary action. It was felt that this fluid impeded the accuracy of the reading. Some men just wash their instruments in hot water.

I have developed a tonometer "sterilizer" that employs ultraviolet radiation-ozone exposure close to the tip. This instrument was made with the cooperation of Dr. L. J. Buttolph and staff at Nela Park, General Electric Lamp Division, Cleveland.

The following data are taken from LD-14, G. E. Ultraviolet Sanitation Booklet, page 6:

We employ a small mercury vapor lamp using only 4 watts. The electrons travel over an arc across a coiled tungsten filament in an atmosphere of mercury vapor. This generates short wave ultraviolet radiation which is efficiently transmitted and emitted through the glass bulb. Ozone is produced within a few inches from the bulb. Ozone is unstable and quickly reverts to ordinary oxygen.

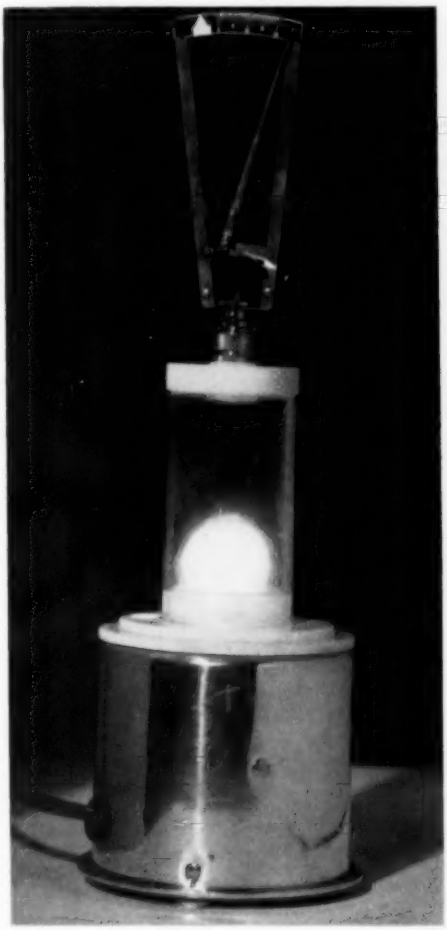
Extensive tests by Dr. M. Luckiesh and associates have proved the germicidal effect of this ultra violet bulb. Its radiant energy kills bacteria, viruses and fungi.

All the organisms found in nebulized saliva were killed in less than one minute when 2 inches from the bulb. This is true also of alpha streptococcus and staph. aureus. E. coli dry and moist were also killed in the same atmosphere in less than one minute. This also applied to beta hemolytic strept. and many molds. 70% Aspergillus niger spores were killed in like manner.

By getting very close to the bulb, the killing of all organisms is greatly increased and the killing time greatly reduced.

It should be noted that Staphylococcus aureus, alpha Streptococcus, Escherichia coli, beta hemolytic Streptococcus, and typical molds and spores are all killed within one-quarter minute when within 1 in. of

Tonometer Sterilizer Stand



the bulb. As an extra precaution, the plunger should be kept in this sanitary atmosphere for longer periods. I have kept the light on all day and have noted no evidence of corrosion on the tonometer.

The rate of germ destruction is directly proportional to the number of organisms and their resistance. It takes longer to kill 1,000,000 bacteria than 100.

The tonometer should be cleaned after each use. No protein should be allowed to coagulate, as a film could thus be formed which might harbor large numbers of organisms. It is known that organic matter reduces the activity of all germicides.

Tonometry should not be done on grossly infected eyes. If it is done, 80% isopropyl alcohol should be used to clean all infected material and the tonometer left in the sanitary atmosphere for a longer time. The electronic tonometer should be cleaned only with dry cotton and pipe cleaners. Alcohol may injure the wiring.

The advantages of this method are that (1) the tonometer is not injured by corrosion; (2) there is no irritation to the eye itself, and (3) the method appeals to the esthetic sense of the ophthalmologist.

This instrument can be obtained from the Storz Instrument Company, 4570 Audubon Ave., St. Louis 10. The stand is made of aluminum.

607 Rose Building (15).

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Registry of Interesting Cases

It is the purpose of this section to record cases on the basis of interest only. No complete review of the literature is attempted and no claim for priority is implied except when specifically stated. Publication of case reports is subject to the concurrence of the Editorial Board on the interesting aspects of the case. It is requested that the reports submitted be as brief as is consistent with inclusion of the pertinent facts, bear a title that is as specific as possible, be prefaced by a paragraph that states why the author feels the case is of interest and be accompanied with photographs whenever possible.

SPONTANEOUS RETINAL DETACHMENT IN FOUR-YEAR-OLD CHILD WITH INTERESTING FAMILY HISTORY

RETINAL detachment in children is not uncommon, but very few cases occurring under the age of 4 years have been reported. Of 682 cases of retinal detachment reported by Arruga [1] in 1936, only 4 patients were under 5 years of age. Pischel * recalls only two cases of detachment of the retina in children under 5 years of age, both associated with high myopia. Above the age of 5 years detachments seem to be much commoner and are frequently associated with a family history of myopia.

The following is a report of a case which presented an interesting family history demonstrating the familial characteristics of this disease.

REPORT OF CASE

J. D., aged 4 years, was first seen in the Eye Clinic at the University Hospitals on Sept. 22, 1954. The child's guardian aunt had noted slight redness of the left eye for a week prior to admission. It was suggested by the aunt that the child might be nearsighted, since the child held objects close to her face when examining them. Both parents were blind, and there was a history of visual difficulties in other members of the family.

Examination.—Right Eye: Va, 3/60, improved to 6/20 with −12.00 sph ⁻ 2.00 cyl, ax 90. The iris was blue. The only other finding of significance was the prominence of choroidal vessels, producing an unusual tessellation of the fundus. Intraocular pressure was normal to fingers.

Left Eye: Va, hand movements at 1 meter. The iris was greenish-blue and the anterior chamber deep. There was iridodonesis. Marked atrophy of the periphery of the iris was noted by indirect illumination. There were many water cysts in the posterior subcapsular area of the lens. The lens appeared subluxated posteriorly but was apparently still within the fossa patellaris. The retina was entirely detached and appeared atrophic and transparent. Intraocular pressure was normal to fingers.

The child was placed on detachment routine with bed rest, and, although she was very cooperative, the retina showed no evidence of settling back. On Oct. 18, 1954, a scleral resection was done on the left eye in the region of the equator and extending halfway around the globe. Postoperatively there was no apparent improvement in the appearance of the retina. Because of the atrophic appearance of the retina, no further surgery was recommended.

Family History.—The patient's father, A. D., wore glasses from the age of 4 because of myopia. At the age of 15 he noticed sudden loss of vision in the right eye. When seen at the University Hospitals one year later, a diagnosis was made of intraocular hemorrhage with secondary glaucoma, O. D., and complete retinal detachment, O. S. Both eyes subsequently became blind.

Of the father's seven siblings, all had good eyes except one brother, W. D. He was born with cleft palate and congenital cataracts. He underwent linear extraction of his cataracts at about 20 years of age, and 5 years later examination showed complete retinal detachment in the right eye. Vision has remained good in the left eye.

The patient's mother, R. J. D., was first seen at the University Hospitals in 1934, at the age of 15. She had worn glasses since the age of 4, with a diagnosis of high myopia. At 11 years of age she suddenly lost vision in the left eye. A diagnosis of retinal detachment was made,

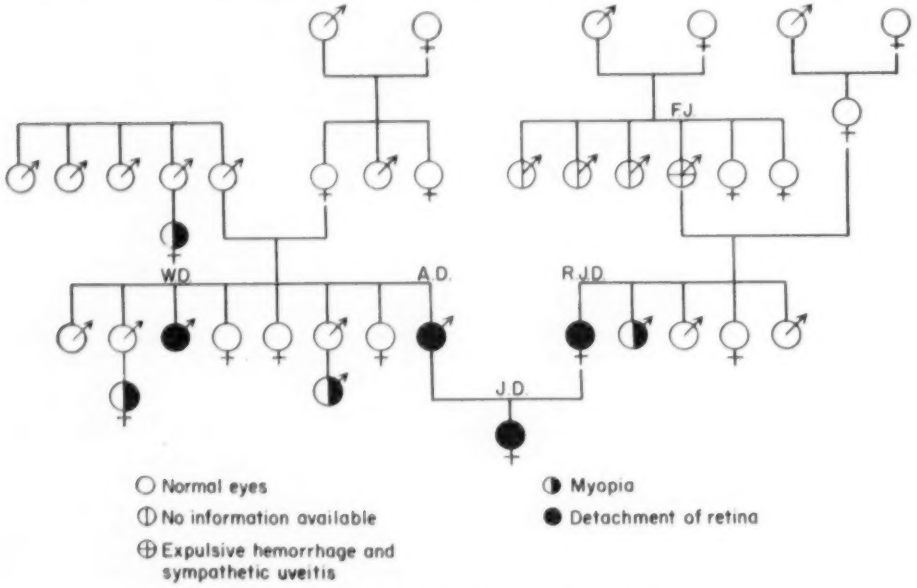
From the Department of Ophthalmology, State University of Iowa College of Medicine.

* Pischel, Dorhmann K.: Personal communication to the authors.

and the eye eventually became painful and was enucleated. When she was last seen, in 1940, she had light perception, O. D., following sudden loss of vision several months earlier, and a dense complicated cataract which obscured the view of the fundus. She was presumed to have a retinal detachment, O. D.

The patient's maternal grandfather, F. J., developed senile cataracts. Following cataract extraction, O. S., there was an expulsive hemorrhage, and the eye was eviscerated. Two years later he developed chronic recurrent plastic iridocyclitis, O. D., and was treated for sympathetic uveitis. Vision was reduced to light perception, and the right eye was enucleated elsewhere about a year later.

The occurrence of blindness in this family can be linked to myopia in most of the cases. Retinal detachment in association with myopia is often secondary to cystic degeneration of the retina at the equator and the ora serrata. Some familial cases of retinal detachment not associated with myopia show equatorial retinal



Pedigree showing spontaneous retinal detachment and myopia.

degeneration and retinoschisis. Schepens † is of the opinion that the familial characteristics of myopia and equatorial degeneration are carried by different genes. It would appear that in the present case these two genes are found together in the same patient.

That degeneration of the retina should occur in some of the descendants of this family was to be expected. That the degeneration should occur at the age of 4 years and progress to complete detachment is unusual. From the history of this family, the detachment of the retina seemed to occur about one decade earlier in each succeeding generation. From this apparent progression, one might suppose that a descendant of the patient whose case is presented here might conceivably show retinal degeneration and detachment before the age of 4 years.

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Landis C. Stewart, M.D.
University Hospitals, Iowa City

† Schepens, Charles L.: Personal communication to the authors.

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Annual Reviews

OPTICS AND VISUAL PHYSIOLOGY

A Review of the Literature for 1954

PAUL W. MILES, M.D., St. Louis

REFRACTION

SLOANE [1] and others bravely report detailed tests of refraction and visual acuity by each of three ophthalmologists on 21 adolescent patients without cycloplegia, performed the same day. The patients were intelligent and cooperative, but in only one case did the final prescription exactly correspond. Sloane concludes that 0.50 D. or so or a difference in axis of 15 degrees for a 0.75 D. cylinder would not be likely to be important in these cases. Perfectionists like the late E. V. L. Brown would take violent exception to this. Users of routine cycloplegic would smile knowingly. Such variations are probably not important unless they induce heterophoria.

The Rodenstock refractometer has been thoroughly tested.* It is agreed that the instrument is speedy and accurate, especially when used with dilated pupils. The authors added that for a master of retinoscopy the refractometer has no advantage. Patients who are difficult subjects for retinoscopy are also a problem for the Rodenstock refractometer.

Two authors † used for a refraction target a 20 cm. circle of tiny luminous dots. Although related to stigmatoscopy, the method was not highly recommended.

The use of "slab-off" prism in glasses for anisometropia is reviewed.[6] For better glasses, one should make careful measurement of the interpupillary distance.[7] Trifocal lenses are praised.‡ The segment position in finished glasses can be checked [10] by inspection of the shadow of the segment border on the patient's eye seen from the aperture of an ophthalmoscope held in the patient's reading position. Glasses and visual requirements for marksmanship are discussed.[11] Ametropia, presbyopia, large pupils, and attempts to shoot from a shady place into the sunlight are the chief handicaps.

The Turville method of binocular refraction is praised.§ By a mirror arrangement, both eyes fuse part of the test target, but only one can see the letters. Patients accept more plus lens binocularly, except when there is exophoria. Another advantage is the ease of testing fixation disparity, which is Ogle's heterophoria test in the presence of fusion. The prism diopters of fixation disparity measured were satisfactory in the glasses of 29 patients, according to two optometrists.‖

The German Ophthalmological Society [16] announces that ophthalmologists cannot relinquish the art of refraction to optometrists because it is necessary to follow the course of ocular disease. Determination of the proper power of lenses is a matter of judgment, like the dosage of drugs. Irvine [17] states that ophthalmologists should not even let it appear possible that they might receive income from dispensing glasses.

There were five cases of diabetic myopia temporarily of about 4.50 D.[18] The anterior chamber became shallower by 0.3 to 0.7 mm., and the lens cortex appeared edematous.

From the Department of Ophthalmology and the Oscar Johnson Institute of the Washington University School of Medicine.

* References 2 and 3.

† References 4 and 5.

‡ References 8 and 9.

§ References 12 and 13.

‖ References 14 and 15.

Digitalis eye drops are helpful in accommodative asthenopia by reducing cholinesterase.[19] Asthenopia due to autonomic dystonia, ciliary spasm, and neuritis may be treated with snake venom (Ammodyn).[20]

ASTIGMATISM

Elaborate corneal studies[21] were made showing that keratometer readings on each side of center became progressively flatter. The pupil seldom centers on the most regular part of the cornea. Peripheral corneal astigmatism may exceed central astigmatism by 2.25 D. Keratometer readings were compared[22] with retinoscopic readings in 100 cases of postoperative aphakia. The keratometer underestimated higher degrees of astigmatism. A similar study[23] compared the astigmatism in 2068 eyes. In the discussion, Cowan states that few use the ophthalmometer routinely, but that it can give valuable information in some cases.

A variable crossed cylinder is reported useful for cylinder retinoscopy,[24] but the author doubts that it will replace the set of fixed crossed cylinders. A new chart for testing astigmatism[25] consists of a 14-in. rotating disc with a grid pattern.

When thyroidectomy is followed by mild proptosis, high astigmatism may develop.[26] In two cases, oblique or with-the-rule astigmatism of 6.00 D. and myopia reduced the corrected visual acuity to about 20/80. The keratograms resembled those in keratoconus except in axis. Temporary astigmatism occurs rarely in pregnancy, and true keratoconus is said to increase in pregnancy, suggesting weakening of elastic tissue by estrogenic hormone. Astigmatism[27] appears in diseases of lowered metabolism as the result of poor corneal nutrition, accompanied by hourglass or spoon-shaped fingernails. Both cornea and fingernails are free of blood vessels.

Corneal-scleral molds and casts[28] were made for eight cases of keratoconus. In the published photographs, it is obvious that the sclera is part of the cone. Comparative photographs of casts of high myopia show a larger corneal diameter and good indentation at the limbus.

MYOPIA

Refractions with atropine were performed on 1182 boys and girls from Poona, India.[29] Except at the age of 12, there were no sex differences. Also reported[30] is the refraction of 590 Brahmin, 618 Chitpavan, and 645 Untouchable children. The last group receives no schooling but has twice the incidence of myopia. Pendse emphasizes the genetic differences more than the possible contribution of dietary differences. No data appeared on exophoria, esophoria, fusion, suppression, complaints, or treatment. Pendse holds that myopia increases during spurts of body growth. In girls, this occurs during the first year of menstruation and may amount to 2.50 D. per year. However, his own figures show that girls are taller and heavier than boys before age 12, and are not more myopic.

Pendse's findings are confirmed by Gardiner[31] from studies of 418 English children. Myopic children were taller and heavier than nonmyopic children, especially at age 14. There was no difference due to sex at age 13, but menstruation began early in the myopic girls. The rate of growth of myopic children was irregular, and frequently the myopes were overweight at age 10 but normal in weight by age 14.

The refraction of 579 children was related[32] to age, change in refraction per year, and amount of refraction. More study was advised of repeated refractions of the same person.

Refraction was done on 508 weavers who worked at a special loom at a fast, fatiguing pace.[33] Thirty-six per cent of the group were myopic, as compared with 9% in the other parts of the factory. Watching the fast-moving machine increased the rate of myopic change. It is urged that myopes be changed to other work and that illumination and working posture be improved.

In the recent war, 30% of Japanese draftees were myopic, as compared with about 20% here. Sato [34] believes that this myopia is due to excessive ciliary tone brought on by near work, and that it should be treated in the earlier stages by mild cycloplegics. Many will prefer this viewpoint to that of Otsuka,[34] who says that myopia is hereditary and not subject to treatment. Myopia is reported [35] in 18,000 Swedish school children. Although of the same age, children in the Latin school showed a 25% incidence of myopia, as compared with 19% in the technical school, suggesting that myopia is proportional to the book work. Lindner [36] repeats his theory that myopia increases because of choroidal hyperemia and hemostasis, which can follow excess near work.

School myopia may be due to defects in orbital shape.[37] In deep orbits, the transverse part of the superior oblique tendon could press on the globe during reading. Five cases of Fuchs's central black spot from myopia are described.[38] One patient was aged 21; 3 were 42 years of age; a fifth was 60. A cyst formed in some cases, causing metamorphopsia. There is stretching, splitting of Bruch's membrane, and tapetal atrophy with migration of pigment.

Full details are reported [39] on a pair of identical twins born in 1906. The boys grew up together, and there was absolutely no genetic or environmental difference to account for one being emmetropic and the other having —25.00 D. or more of myopia. Their keratometer readings were nearly equal.

Although the lamellar scleral resection does permanently shorten the globe, after operating on 71 eyes, Shapland [40] would not advise it in uncomplicated myopia. The optics of the myopic and the hypermetropic eye were studied.[41] There was an increase in hypermetropia and a decrease in myopia of the anterior and posterior angle alpha as determined by Nordenson's technique.

In growing children, the dominant eye usually becomes more myopic than the recessive eye. In strabismus, the dominant eye is less hypermetropic. A haploscopic comparison of the accommodative tone of the two eyes was made.[42] In some subjects the dominant eye accommodates more than the other regardless of the direction of gaze, suggesting that ciliary tone is a factor in developmental dioptric eye changes.

OPHTHALMIC LENSES

Plastic lenses can be nearly as hard as crown glass.[43] They are light in weight, do not break, and do not fog. Corneal contact lenses may slip off in cases of keratoconus.[44] Of all types, the corneal-scleral fluidless lacrimal ventilated contact lenses are most comfortable.¶ Sattler's veil, corneal erosions, and edema caused failure in about 20% of 365 patients fitted.[47] Other clinical reports were published.# Contact lenses can be made without molding by means of stereophotography.[53] Contact lenses for use by "frog-men" under water were designed.[54]

The use of telescopic lenses has been sadly neglected.[55] Not many partially sighted patients have the intelligence and persistence to master them, but test sets should be available for demonstration in the office. In many cases, all that is necessary is a strong convex spectacle lens [56] for reading at 3 or 4 in. For many patients, this produces a better visual field and depth of focus, without the expense. If strong lenses are to be used binocularly, suitable prism must be added. Patients under the age of 40 with visual acuity of 20/200 or 10/200 will benefit from the Feinbloom 6- or 8-power microscopic spectacle.[57] Vision at about 3 in. is somewhat superior to that obtained with 10 to 20 D. of excess plus sphere. The optics of magnifying devices like the Beebe loupe are discussed.[58]

Aniseikonia is discussed from a clinical standpoint.* Magnification formulae are explained.†

¶ References 45 and 46.

References 48 through 52.

* References 59 and 60.

† References 61 and 62.

ACCOMMODATION AND CONVERGENCE

Haploscopic studies of accommodation and convergence in normal and presbyopic eyes [63] indicate that no more effort is required for the last diopter of accommodation than the first, whatever the amplitude available. In presbyopia, accommodative effort must relax because convergence does.

A myope aged 20 is said to have about twice the accommodative amplitude of his hypermetropic brother.[64] At the age of 50, the hypermetrope requires a much stronger reading addition, partly because of lens effectivity at 12 mm. from the cornea.

A case of accommodative paralysis from lead is reported.[65] Curiously, the accommodation was better for a period of about one hour after a meal.

Through an artificial pupil,‡ there was an increase in accommodative amplitude and speed upon increasing illumination up to 1000 lux. Increasing the size of the Landolt ring target also shortened accommodation time. By photographing human Purkinje reflections, it was shown [68] in night myopia that 0.80 D. is due to accommodative tone. Apparently, the human eye accommodates in empty fog or space in light adaptation about the same amount, just for lack of a fixation object. The minimum quantity of light required to elicit accommodation in the dark-adapted eye was 1 millilambert on a 1-degree target. This is about twice the foveal cone threshold, far higher than rod threshold. Therefore, foveal cones are required. At such low intensity, a color fringe would not be likely to be involved in the stimulus. This refers to what Weale [69] calls the most dramatic discovery of the year: Fincham's color-fringe theory of accommodation.

After removal of the upper cervical ganglion [70] on one side in the cat, myelinated fibers to the ciliary body degenerated within 72 hours. After coagulation of the Gasserian ganglion on one side, there was Wallerian degeneration of nonmyelinated fibers in the ciliary body. There are, therefore, two types of nerve supply. Marg [71] stimulated the ciliary ganglion of anesthetized cats and observed the accommodation with a Rodenstock refractometer. With between 0 and 5 volts of oscillating electricity, the linear accommodation suggested a multiunit smooth muscle, in which the action depends on the number of units responding. Lens capsule and fibers were observed under the phase-contrast microscope while applying electric stimuli.[72] There was contraction, but the time was too short for the accommodation to be considered lens-induced.

Some patients will accept +0.50 D. binocularly, a lens which will blur the vision monocularly.[73] This may be due to sympathetic ciliary innervation for negative, or distance, accommodation. In routine refraction the final sphere should be determined binocularly.

In 90% of refraction cases, decentration of the lenses or prescription of prism base in to relax convergence for near work is advised.[74] An optometrist [75] claims success in treatment of 103 from a group of 155 patients with exophoria or exotropia. His prescription of excess minus power of from 3.00 to 5.00 D. is said to cause little discomfort.

As a routine cycloplegic,[76] 1% cyclopentolate (Cyclogyl) is comparable to atropine in Negro eyes. No local or systemic toxic effects were encountered. Cyclopentolate for lightly pigmented irides may be used in 0.5% strength. When methylcellulose solution is used as a vehicle for cycloplegics, only 1 drop need be given.[77]

VISUAL FATIGUE

One of the most fatiguing procedures is to continue testing visual acuity on Landolt rings.[78] After 60 minutes the visual acuity actually decreased 18% to 32% in all subjects. Flicker fusion frequency decreased

‡ References 66 and 67.

about 10% during the hour. This seems to the reviewer a good excuse for rapid methods of refraction. If refraction and motility cannot be tested in 20 minutes, the examination should be continued on another day.

It is not accommodation, but convergence, which becomes fatigued.[79] Technicians operating stereophotographic apparatus become fatigued because accommodation is dissociated from convergence.[80] The psychosomatic approach should be taken in clinical work [81] for better interpretation of fatigue.

TORSION

Again this year, papers have appeared on ocular torsion, only to be refuted. The confusion seems due to terminology, to misleading results of after-image tests without an eye-centered spheric coordinate system, and to poor visualization of Listing's plane. Surely, both eyes make involuntary synchronous or fusional torsion movements, but not necessarily in oblique gaze. Bielschowsky and Javal described the righting eye movement on sidewise tilt of the head. The excyclophoria of upward gaze is accepted.[82] This may be physiological overaction of the inferior oblique muscles.

The after-image method was used [83] to measure the correcting rolling reflex resulting from tilting the body sideways 0, 10, and 45 degrees. The reflex did not occur in a patient with a defective vestibular system. A head tilt of 30 degrees produced compensating rotation on the visual axes of 10 degrees.[84] In 25 subjects whose glasses corrected 1 D. or more of astigmatism, tilting the head caused monocular blurring.

Pascal [85] defines ocular torsion as rotation on the anterior-posterior axis. "True" torsion would be in the same direction for both cornea and retina. In "false" torsion the globe rotates not on an anterior-posterior axis, but on a line in Listing's plane, which is a frontal plane through the center of the globe. In "false" torsion the cornea and retina make a twisting motion opposite each other on the anterior-posterior axis. This simple statement is refuted by Roelofs,[86] who also objects to the term "false" torsion. Raubitschek [87] upholds Pascal in a reply criticizing Roelofs' views.

In a paper favorably discussed by Adler, Quereau § presents helpful illustrations about ocular torsion, confirming Donders, Ogle, Pascal, etc. Hewitt,[91] who wrote about torsion in 1951, complained that torsion should be defined not in a spheric coordinate system, but in terms of the position of the head. Hewitt based his argument on experiments with after-images, while Quereau depended on the change in angle of the line between a limbal vessel and the corneal center.

OPTOMOTOR REFLEXES

The ophthalmograph was improved by a special camera and time marker.[92] When an object fixed by both eyes is moved, there is a delay of 120 to 180 msec. before the eyes turn. The eyes move as though both were stimulated from the central nervous system at the same instant. The acceleration and deceleration are rapid and precise. The following eye movements jerk, as predicted by Ludvigh's feed-back proprioception theory.

Other methods and results of tracing eye movements are described.‖ The time consumed in eye movements or in a reflex blink is vital in flying modern planes. Authorities ¶ warn that during a blink, an approaching plane could collide before an avoiding hand or foot motion could be accomplished. Visual perception time can be shortened by caffeine or pentylenetetrazol, or lengthened by alcohol or barbiturate.[101] The impulse latency [102] from the retina to the geniculate body in man is 30 msec., plus 10 msec. to the cortex. From stimulus to an eye movement requires 100 msec. The time between stimulus and a finger switch response increases in low-target intensity and decreases in high.[103]

Time and other characteristics of autokinetic eye movements were studied.# With the ocular muscles paralyzed by curare and

§ References 88, 89, and 90.

‖ References 93 through 97.

¶ References 98 through 100.

References 95 through 97; 104.

decamethonium,[105] there was no false movement of fixed objects or autokinetic sensation when the subject's eyes were moved manually by the experimenter. Only when the eye moves under its own power can one see autokinetic movement.

Alternating hyperphoria in a series of 113 patients of all ages from the Amsterdam motility clinic were studied.[106] It was not affected by direction of gaze, and the movements were slow and gliding, like fusional movements. Alternating hyperphoria was explained as a disturbance of monocular optomotor reflexes, corresponding to some of the ideas of Keiner. Stimulation of the labyrinth by cold or warm water, or by 90-degree inclination of the head, did not change alternating hyperphoria.[107]

Ten Doesschate[108] observed wide searching nystagmus, like that in congenital amblyopia, when he placed two +20.00 D. lenses in a trial frame before a normal eye at a vertex distance to place the focus in the rotation center of the globe. Eye movements would not change the position of the blurred image on the retina; so the nystagmus may be an exaggeration of normal fixation nystagmus.

READING

Both the phonetic and the flash card system should be used for teaching reading to about 20% of the pupils.[109] Children with aphasia need to learn by use of the ears and fingers, as well as by the eyes. Pathological studies in a patient who suddenly lost the ability to read showed normal visual pathways.[110] There were lesions at the base of the left cerebral hemisphere where the fusiform lobule had disappeared.

Vocalization is one cause of slow reading habits,[111] but the speed of word recognition depends partly on experience in vocalization of that particular word. Words appearing more frequently in the literature are recognized in shorter tachistoscopic exposures.[112]

The determination of visual fixation by the acuity gradient of paramacular cones is discussed.[113] Most of a printed word is imaged on the paramacula, and when recognition requires but one fixation, that is usually in the center of the word. Hemianopsia interferes with reading because half the word is not visible. Macular sparing may follow an adaptation process by which the fixation cone is changed toward the functioning half of the retina.

Children with an amblyopic eye may have reading difficulties[114] when the blind eye is on the same side as the dominant hand. It may be easier to change ocular dominance than hand dominance. Attempts to change cerebral dominance have been futile.

FUSION AND DOMINANCE

The new thinking on the mechanism of fusion started by Verhoeff gained momentum this year. By Hamburger's method, normal subjects were tested under stereoscopic rivalry conditions.[115] Alternations occurred at a rate of 20 to 30 per minute. The alternation rivalry may be in nonfusing colors like red and green, or black and white, or may be rivalry in nonfusing patterns. Inhibition and summation contrast phenomena can be observed where lines or colors superimpose. Persons with more rapid alternation perform better with less fatigue certain tasks like stereoscopic map making and stereo-range finding. The rate of alternation correlates with retinal adaptation.[115] In a few seconds, color contrast and pattern in one retinal area fades, when the other, corresponding retinal area must become dominant to maintain a constant cerebral image. A strong retinal inhibition reflex for the corresponding point fades with the local adaptation, and the inhibition stops. In lower animals, alternation must occur, or there would be diplopia.

The observation by Hering and other early writers that normal eyes alternate while reading was confirmed.* I can see this easily by reading with red-green glasses. Any anomaly which would make the vision or the movement of one eye inferior would interfere with

* References 115 and 116.

rapid alternation, and would therefore impair stereopsis. Interference could follow a subtle change in dominance of nasal over temporal retina.

Of two equidistant objects, a person perceives that one as nearer which is homolateral to the dominant eye.[117] The dominance could be varied or alternated by neutral density filters or fogging lenses. Normally, foveal dominance is equal bilaterally. Upon tests of the dominance of areas of the binocular visual fields toward the right of fixation, the right retina becomes gradually more dominant until the monocular crescent is reached. In spontaneous anisodominance, central dominance favors one eye a percentage which is distributed equally over the binocular field. This causes an error in relative distance perception of mass objects in space. Anisodominance differs from anisopia and from aniseikonia in not requiring stereopsis. Anisodominance, like anisopia, can be due to anisocoria, damage to one retinal image, or a temporary change, such as unilateral dark adaptation.

Suppression is the chief factor in the new alternation theory of fusion,[118] according to ingenious experiments. There can be no fusion in the traditional sense, because there is no doubling of brightness or visual acuity.

However, binocular vision is slightly better than monocular vision in both brightness and acuity. This binocular improvement was observed[119] in spite of plus sphere fog intended to prevent the well-known effect of binocular vision to lower the accommodative level. The improved binocular visual acuity can be attributed[120] to an impairment of a monocular foveal image by the asymmetry of the light ray bundle. Since the visual line is not on the geometric anterior-posterior axis of the globe, lens, and cornea, it would be a strange coincidence if the foveal light rays arrived symmetrically or perpendicularly. The asymmetry would make the foveal image superior on one side for one eye and on the other side for the other eye. Only by combining the better parts of the two retinal images could the visual cortex obtain the best possible picture. Here Verhoeff's theory gains evidence. He said that the cortex selected and held any part of either retinal image it found most useful.

In Sherrington's classic binocular flicker studies, the fusion rate for monocular vision was 3% less than that with alternate flashes seen by the other eye. With more precise electronic equipment, Perrin[121] found binocular flicker 8% higher than monocular. Shikano[122] found little evidence for such binocular cooperation in improving flicker fusion rate.

Charnwood[123] presents stereoscopic experiments which seem to contradict some of the new alternation theories. The matter is not as simple as many writers seem to believe, for their stereoscopic illustrations are too crude. Stereopsis is a miracle of precision, and it is almost impossible to set up fusion contours exact enough to control stereopsis. I can see physiological diplopia in nearly all of the published stereograms. Only corresponding points are said to alternate, not those in physiological diplopia.

The difference in sensitivity of temporal retina to length discrimination as compared with nasal retina was studied.[124] The difference was considerable, but it gradually disappeared with age. More research is indicated, because the difference may be related to ocular motility problems of children.

The dominance of peripheral fusion over macular under certain conditions is discussed.[125] Optomotor reflexes from peripheral retina may dominate those from the macula. Size and contrast are important parts of a peripheral stimulus, while color and form attract attention in the macula.

The fusional amplitude was well measured[126] by the duration between flash exposures in a haploscope which will just permit fusion against a certain angular stress. Experiments were performed on vertical and cyclofusional movements.† There is torsional, as well as horizontal and vertical, fixation disparity.

† References 127 and 128.

PERCEPTION LATENCY

The Pulfrich phenomenon, in which a pendulum swinging in a frontal plane appears to describe a horizontal ellipse, is due to the lag in perception time in one eye produced by a gray density filter. The lag was calculated in milliseconds [129] for various filter densities. Darker filters soon interfere with stereopsis and stop the experiment. Other experiments were presented in which one target in a stereoscope was printed partly on a gray background.

The Pulfrich phenomenon can occur under conditions eliminating convergence and accommodation.[130] What resembled the Pulfrich phenomenon can be produced [131] by placing a prism before one eye. This might appear to interject convergence as a cause of the phenomenon except for the fact that a flat prism produces stereodisparity. The power of a prism varies with the angle of incidence.

Perception latency multiplied by the disparity in the Pulfrich phenomenon is a constant.[132] There is a flattening of the near part of the elliptic path due to monocular depth factors, such as brightness.[133] This seems reasonable, because the ellipse is best seen on peripheral retina, in which brightness is more important. Reducing the light intensity for one retina has a greater effect on perception latency in peripheral than in macular retina.

Perception latency may be measured by a combination of rotating discs.[134] The increase in time was greater from a density filter than could be predicted by the Pulfrich phenomenon.

STEREOPSIS

The Luneburg theory that visual space can be described by Riemannian hyperbolic geometry has been confirmed in subjective studies by the late L. H. Hardy and associates.[135] In 68 pages, they show how to express the subject's binocular metric space perception as a number. It is a refined test of stereofunction, as well as a description of individual differences in visual localization and orientation.

Ogle ‡ objects to the common use of the term "stereopsis" when the writer means to say "depth perception." He cites a shameful error being made by some of the gestalt psychologists: "The theory of local depth signs of disparately stimulated retinal elements cannot be upheld . . . as far as optics are concerned." Ogle, of course, provides irrefutable evidence. Wallach,[138] for instance, made shadow images of a three-dimensional wire object. As long as the object remained stationary, the shadow on the screen appeared two-dimensional. However, when the wire figure was rotated slowly, the image on the screen appeared in three dimensions. Since no disparity could possibly be present, he concludes that stereopsis does not require disparity. This phenomenon can be observed monocularly; so Wallach has no right to use the term "stereopsis." How do gestaltists explain the fact that stereoscopic acuity and the distance limits of stereopsis are proportional to the interpupillary distance?

To adjust stereoscopic disparity for perfect depth value and also comfort in three-dimensional movies requires experience.§ Large close-ups require less disparity. Most of the discomfort reported is heterophoria or difficulty in adjusting the convergence required to the absence of accommodation required. Stereomovies are said to be excellent for fusion training.[142] The new Polaroid television viewer does not require stereopsis, but should prove helpful in fusion and amblyopia training if the child's head stays erect.

There is an excellent review of the phylogenetic development of binocular vision.[143] There is also a review of the anatomy and physiology of the chiasm and geniculate bodies in animals and primates.[144]

DEPTH PERCEPTION

The Thouless effect is an illusion in which a plane disc or ellipse tilted off the frontal plane appears erroneously to be more nearly in a frontal plane. This may be experience,

‡ References 136 and 137.

§ References 139 to 141.

not depth perception.[145] The discrimination of position of a luminous Thouless disc is better in the dark, which interferes with the illusion.‖

The psychologists ¶ report tests of the apparent midsagittal plane or the apparent vertical in normal subjects and in patients with brain disease.

There is an illusion noticed in dog or horse races in which judges on each side of the finish line tend to see the most distant animal pass the finish line first.[152] Possibly one should watch this in automobile traffic, especially if a policeman is around.

Of the visual clues involved in flying and landing an airplane, linear perspective appears to be most reliable.[153] Next in order are retinal image size, stereopsis, and motion parallax. The increase in density of patterns on the terrain with distance, which Gibson emphasized in his book on perception, was most important. Landing an airplane on a homogeneous surface would be dangerous. Flying ability improves the training in perspective discrimination.

Some subjects made good scores in the Howard-Dolman test who did poorly on the Verhoeff Stereopter.[154] The former test introduces undesirable monocular clues and depends partly on aniseikonia and the horopter position. In the three-rod stereoacuity test # depth perception of older persons could be improved by dilating the pupils, while that of myopes could be improved by excess minus lens. Heterophoria appeared to interfere with depth perception more in daylight than in the dark. In 1016 subjects, the best depth perception was found when the middle of the three rods conformed to the shape of the horopter with all three in Panum's fusional areas.

Size constancy and related perception received attention.* An interesting size phenomenon might have clinical application.[159] When a person looks at a wall 27 in. away through a circular 1/4-in. aperture 3 in. from the eye, a large blur circle fluctuates irregularly with variations in pupil size.

‖ References 146 and 147.

¶ References 148 through 151.

References 155 and 156.

* References 157 and 158.

INTRARETINAL REFLEXES

Experiments show that stimulation of a retinal area decreases the sensitivity of adjacent, and even remote, areas of the same retina. Boynton [160] challenged the idea that this is reflex action. Part of the change in sensitivity could be due to the veiling effect of stray light. Fry and Alpern † state that the metacontrast phenomena involve both stray light and inhibition reflexes. Metacontrast was measured from flash luminances of from 1 to 1000 foot-lamberts.

In studies of foveal summation, it was found [163] that red- and blue-sensitive receptors behave differently at threshold when luminance and area are varied. A red patch needs more luminance to appear equally as bright as the same area of blue. The law that area times intensity is a constant at threshold appears related to the proportion of cones to nerve fibers.[164] Tests of foveal summation in a patient with amblyopia ex anopsia [165] suggest local impairment of brightness discrimination and intraretinal reflexes, a finding which is against the cortical suppression theory.

GLARE

Tinted glass in automobile windshields is dangerous for night driving. The visual acuity of patients with and without sensitivity to glare under simulated night-driving conditions against oncoming headlight was tested.[166] Measured by the Clason acuity meter, average vision through colorless glass was 20/32. This decreased to 20/34 through light-yellow lenses, to 20/40 through ordinary second-shade pink glasses, to 20/46 through green windshield glass alone, and to 20/60 through the green windshield and pink glasses combined. The acuity was worse in those with sensitivity to glare. Subjects in the habit of wearing pink-tinted glasses had better than average acuity in that part of the

† References 161 and 162.

test. The visibility distance in night driving [167] was reduced by tinted glass 12% at 2000 ft., and 45% at 100 ft., distance. Resolving power, stereopsis, discrimination of angular velocity, simultaneous contrast, etc., are all decreased by tinted glass.

Amber light is in common use to penetrate fog. Under simulated fog-driving conditions, there was "no evidence of any improvement in seeing . . . with yellow light when compared with a white beam of equal intensity."[167] Yellow glasses may improve chromatic aberration and stray light in the eye, but do not improve the visual acuity.[168]

Ocular stray light is very important.[169] It interferes considerably with vision, and is counteracted partly by the Stiles-Crawford directional effect. The electroretinogram evidently comes from stray light, because it makes no difference whether the stimulus is directed to the fovea, to the periphery, or to the disc. Why glare is worst at an angle with the visual line of 7 to 8 degrees cannot now be explained. Techniques for isolating the effects of stray and focused light are described.‡

VISUAL ACUITY

An international visual acuity chart is proposed [172] in which the unit of measurement is the "snellen." The geometric progression of the letters would be the same as on the present Lancaster card. Snellens from 2 to 100 would be determined by formula: 50 times the actual distance divided by the distance a line should be read by the normal eye.

To answer criticism about the reliability of the Orthorater, Gordon [173] made photographic reproductions of the targets to be projected on a screen. The instrument and its replica at distance were compared on 117 subjects. Retests after two weeks showed greater repeatability in the instrument. Krimsky [174] opposes these instruments vigorously.

> Any method which hides the eyes and renders inspection impossible is often unreliable. This is true not only of the phorometer but especially so of the new fangled glamorized version of the old hand stereoscope, and is known as the Orthorater and the Sight Screener, which is of absolutely no clinical value.

Objective tests of visual acuity by the method of optokinetic nystagmus are fully documented.[175] A drum fitted with rings of checkerboard pattern in various sizes, to be exposed one at a time, rotates from 3.5 to 15 turns per second.[176] If the pattern is large enough to be discriminated, the eyes make involuntary nystagmus. The method can be applied to infants, animals, and even insects.

Tests of visual acuity in children should be made at a reduced distance.[177] An ordinary music metronome can be used [178] to test the visual acuity of infants. The pendulum swings 20 times a minute, and is fitted with different-sized discs, which determine the acuity by attracting following movements.

Habits of visual acuity and fusion become fixed after the age of 8 years.[179] The referral of school children to ophthalmologists is discussed by questionnaires.[180] Of the New England doctors replying, 62.9% thought a school nurse could be taught to make routine tests for hypermetropia. As much as +1.75 D. of hypermetropia was considered insignificant in the absence of heterophoria. The limits of heterophoria calling for referral were placed at 6△ horizontal and 2△ vertical. Many of the ophthalmologists tested color vision and stereopsis routinely in children.

The blurring of vision in multiple sclerosis is partly due to oscillopsia, which decreases in oblique gaze.[181] Deaf children were below average in the ability to construct figures from visual models and had trouble in distinguishing foreground from background.[182]

In a symposium on visual requirements in relation to modern travel, Duke-Elder [183] points to some psychological causes for accident. Once a pilot has learned to fly, he can do without stereopsis, and can even fly safely without foveal vision.[152] In high-altitude flight or in fog where no object exists to attract fixation, the eyes involuntarily accommodate enough to blur, and fail to see an

‡ References 170 and 171.

object when it does appear. Under such conditions, glasses with an excess —0.75 D. sphere should be worn.

The distortion from curved windshields is noticeable, but not dangerous.[184] Perhaps more important is vibration. The vibration of a train was found to be approximately 1.5 mm. at 5 cycles per second. This is enough to blur vision, cause fatigue, and reduce reading rate 5%.

Raising the color temperature of an object increases its apparent brightness except in older persons.[185] The brightness of illumination appears to increase when it flickers off and on 9 to 15 times a second, but this "Bartley effect" will not improve reading under restricted intensity.[186] An automatic tape recorder makes each stimulus and records the response for visual experiments.[187]

VISION MECHANISMS

Of those investigators now revealing the intimate physical and chemical details of rod and cone vision, Wald § is famous. A greater light stimulus increases the percentage of rhodopsin bleached surprisingly little, while it lowers the visual sensitivity markedly. In about 1400 layers in each rod are 18,000,000 moles of rhodopsin, each of which can discharge a rod on contact of 1 quantum of light. If 100 of these layers absorb 1 quantum of light each, the threshold would have risen above absolute threshold about 1400/1400 —100 times. While 1 quantum has a chance to discharge a rod, subsequent additional quanta have the chance of being absorbed uselessly by layers having already discharged.

The rhodopsin-digitonin complex is discussed,[190] and the theoretical importance of the dichroic nature of rods, observed by Schmidt in 1938, is cited.[191] Spontaneous excitation in rods and cones (noise) is negligible, making vision a stable and repeatable phenomenon at threshold.[192] At the absolute threshold, so small a percentage of the incoming photons can be used that the sensitivity maximums are set by the "noise" limitations of the incoming photon stream.[193] An apparatus to demonstrate quantal fluctuation in vision is described.[194] A "decision-making" theory of visual detection is announced.[195] Others have shown that at threshold, better data on vision can be obtained by the forced choice—yes or no.

Various experiments with the Stiles-Crawford directional sensitivity and the detection of the plane of polarized light by the simple Limulus eye were made.[196] A magnetic field can stimulate a weak diffuse visual sensation,[197] but nothing in the literature suggests that a magnetic device could by-pass an opaque cornea with a useful image.

A method of testing human visual sensitivity by induction of electrical phosphenes ‖ is described. Applied through electrodes anywhere on the body, 20 cps will produce the most sensation for the least stimulus in the light-adapted normal eye.

COLOR VISION

The new Hardy-Rand-Rittler Polychromatic Plates ¶ were compared with the Nagel anomaloscope, the Ishihara plates, and Farnsworth's Dichotomous Test. Of 150 color-defective subjects, the Nagel instrument scored 98%, the H-R-R Plates 97%, and the Ishihara plates 74%. Routine color tests should be done on school children at the age of 13.[202] In a group of 1000 Batutsi and 1000 Bahutu natives tested on the Ishihara plates, only 2.5% had color deficiencies.[203] Mentally disturbed patients may fail isochromatic color tests temporarily.[204]

Seasonal variations in color vision were explained by variations in vitamin C intake.[205] Color vision improves at an altitude of 2000 to 3000 meters,[206] but then decreases due to anoxia. Older people lose some color sensitivity except for red.[207] Minor blue-yellow blindness is probably not sex-linked.[208] Genetic studies suggest four retinal color receptors.

§ References 188 and 189.

‖ References 198 and 199.

¶ References 200 and 201.

Two sets of three laminae each in the lateral geniculate body appear not to have any color function on the basis of studies of the monkey and the cat.[209] The cat is completely color-blind [210] but has well-developed laminae.

Persons vary considerably in ability to match and differentiate colors.# A violet receptor has been identified in human color vision.[214] Dark-adaptation curves were taken after exposure of the eye to one of the various colors. When a violet-stimulus threshold target was used, a sensitivity peak occurred in two to three minutes' adaptation, which was present in both protanopes and deuteranopes. Adaptation to a given hue decreases the luminosity curve in the part of the spectrum represented by that hue.[215] Red and green retinal mechanisms were isolated by testing visual acuity of colored gratings on a background of another color.[216] Blue could be distinguished from rod thresholds. The red and green receptors may use the midget bipolar and midget ganglion cells in the retina, while blue receptors discharge through flat or brush bipolars. Visual acuity in blue light is less than that in equal red or green light.

The Fechner patterns are attracting the attention of psychiatrists as a by-product of photic driving in diagnosis and treatment. Burnham [217] said the dramatic, unearthly bright-colored patterns were best at 40 flashes per second and disappear in monocular vision. With either the sector disc or the electronic flash photo-driving apparatus, Ulett and I cannot confirm this. The Fechner colors and patterns appear best between 9 and 16 flashes per second. Occlusion of one eye completely will change the pattern and color, but will not erase it.

Color sensation is partly stimulation and partly inhibition or masking.[218] Color and brightness are qualities of the same receptor, but take different paths to the cortex. While light on a target is changed gradually from red to green, the sensation is abrupt, due to the sudden shift to red inhibition. A similar change from blue to green does not give the sudden subjective difference because blue excites the red receptors, which, in turn, inhibit the green.

Total color blindness may be retrobulbar,[59] since red sensitivity is higher in cone monochromats than in protanopes, and in view of the Fincham color-fringe accommodation experiments. Normal eyes accommodated more for a target illuminated by a yellow consisting of mixed red and green than for a pure yellow. The subjective color sensation was identical. Three total cone monochromats who were tested showed the same reaction, although red-green blind subjects were quite different.

Many cases of congenital hereditary achromatopsia are reported.* These patients can match colors only in the gray series. They have poor central vision, which improves in dim illumination. There is no Purkinje shift. There is photophobia, a slow pupillary light reflex, and a fine pendular horizontal nystagmus, which disappears gradually by the age of 30. The fovea is absent or defective.

FOVEAL ENTOPTIC PHENOMENA

Entoptic phenomena which fascinated the early writers are again exciting attention. W. R. Miles [222] quotes Maxwell on his discovery of his spot in 1856: "The most convenient method of observing the spot is by presenting the eye in not too rapid succession, blue and yellow glasses." By alternation of a dark-purple and a neutral gray filter, the entoptic spot provides a measure of foveal size. Of 20 normal observers, 19 were able to see Maxwell's spot, but it is quite subjective and variable.

The Haidinger brush phenomenon is discussed [223] and theories developed involving polarized macular pigment and local adaptation. Tests of Maxwell's spot and Haidinger brushes were made in 45 patients with glaucoma.[224] Evidently a central visual acuity of 20/200 with best correction or better is re-

References 211 through 213.

* References 219 through 221.

quired for these phenomena. A person with cataract must see 6/30 or better to be capable of observing Haidinger brushes.[225] To see Maxwell's spot, one must have good red-green vision. Children will not see the phenomena unless they have unusual powers of observation. Minimal macular conditions, like edema, hemorrhage, exudate, or cyst, will interfere.

PERIPHERAL VISION

A fresh approach in visual field testing comes from an English neurologist.[226] Peripheral retina was tested with tachistoscopic exposures of two white targets against black, to discriminate "one" or "two." He also found that testing retinal adaptation time with a stopwatch would frequently give valuable information out to about 30 degrees.

The effect on visual fields of corrective spectacle lenses was less than expected.[227] The increase in retinal image size without glasses may partly neutralize the decrease in brightness and contrast. An objective method of perimetry was described,[228] using optokinetic nystagmus.

Visual field screening tests were considered.[229] The pattern is exposed for a moment, and an incomplete description from the patient indicates further testing. Walls[230] warns that defects in perception of a pattern may not be detected because of "filling in." Some persons refused to believe Mariotte when he first described the blind spot, because when they looked at a horizontal wire, they could see no break where the blind spot should be in monocular vision.

DARK ADAPTATION

Three new methods of testing dark adaptation were described this year. One has targets illuminated by radium paint, in which the patient is to arrange small cylinders in order of their brightness.[231] A device for testing soldiers is practically self-operating.[232] The subject sees gray contrast patterns and turns a handle to keep them equally bright as they are exposed 0.1 second each two seconds during dark adaptation. The position of the handle prints the curve on a rotating drum. The projection adaptometer of Rieken-Meesman is described.[233]

The reliability of dark-adaptation tests in the Hecht-Schlaer instrument was within 4 log units for two subjects tested repeatedly for 11 months.† Dark adaptation is not only chemical but nervous,[236] since it depends on the size of foveal targets at threshold. This summation effect was confirmed by others.‡ Dark adaptation is more rapid in complete darkness than in gradually increasing darkness.[239] Experiments show interaction and summation of subliminal retinal stimuli grouped at intervals in time and space.[240] The relation of intensity discrimination thresholds, the ERG b-wave, and dark adaptation is discussed.[241]

Dark adaptation can be improved by intramuscular carcholin.[242] The law that area times intensity for threshold stimuli remains a constant applies also to peripheral retina in dark adaptation.[243]

FLICKER FUSION THRESHOLDS

Macular flicker fusion tests were made in patients with glaucoma, arteriosclerosis, retrobulbar neuritis, and the like. Nerve defects appeared to reduce flicker fusion rates more than retinal lesions.[244]

A comparison of flicker fusion fields with standard field technique was made in 90 patients.[245] The flicker fusion method was found more revealing in 12 cases, equal in 66 cases, and inferior in 12 cases. The flicker target excelled for comparing nasal with temporal retinal sensitivity, but might miss a small scotoma or small notch in an isopter. The authors found poor repeatability outside the 30-degree circle. Better results were obtained by Weekers in the region outside 30 degrees by enlarging the target for that part of the field.

The flicker fusion threshold is the "most reliable neurological test to detect the acute effect of a dose of barbiturate."[246] The flicker

† References 234 and 235.

‡ References 237 and 238.

fusion rate decreases about 10%. Flicker fusion perimetry is a useful office procedure in patients with glaucoma and neuro-ophthalmological diseases.[247]

A flicker instrument can be built for about $65,[248] but the Strobotac is the only accurate and permanent device I have seen which is suitable for office use. Some projection perimeters can be adapted for flicker fusion.[249] The 1-meter wire perimeter works very well.[250] The flicker end-point can be checked with an oscilloscope.[250]

Flicker fusion thresholds can be used to predict the severity of eclampsia.[251] Smoking a cigarette will lower the flicker fusion rate in most subjects.[252] Alcohol and hypoxia together reduce flicker fusion rate more than either alone.[253] Some persons show a small diurnal variation in flicker fusion rate which depends on the basal metabolic rate.[254] Landis [255] has reviewed physiological studies of flicker fusion phenomena.

ELECTRORETINOGRAPHY

A great deal of attention has been given to electroretinography, and it may develop into a valuable objective test of visual function. Many clinical studies of the ERG were made.§ At present, it is disconcerting to find a normal ERG in a blind eye, as occurs in diseases central to the rod and cone layer. On the other hand, the ERG may be extinguished in a functioning eye in early retinitis pigmentosa. Diseases which interfere with the nutrition depress the ERG.

For physiological studies, the ERG and electroencephalography proved a valuable measure of the time between a stimulus, retinal response, geniculate response, and cortical response.[263] In congenital total color blindness, there was absence of the x-wave, a diminished a-wave, and a moderately diminished b-wave.‖ Goto and Toida [266] thought a series of ripples in the d-wave indicated color response, but Armington [267] refuted any such idea. Interesting results were obtained by exposing the macula to one color, then making an ERG from a flash of another color.¶ Adaptation to red fatigued the cones and reduced the x-wave, while adaptation to blue fatigued rods and reduced the b-wave.

Typical dark-adaptation curves can be made using the b-wave amplitude.# Flicker fusion tests are valuable in driving the ERG.[272] Some use an oscilloscope and kymograph camera for the ERG.[273] This is more sensitive than the ink-writing encephalograph, which most men are using, but it is less convenient. Burian [274] improved the contact lens electrode and reports [275] a depression of the alpha rhythm upon stimulation of an amblyopic eye. There was weaker photic driving when the amblyopic eye was stimulated than when its normal fellow was stimuated.

Animals treated with sodium iodate or sodium azide show selective destruction of the rods, cones, and pigment epithelium.[276] Results in the ERG indicated that a- and b-waves originate from the inner retinal layers, including the outer plexiform layer. The c-wave originates in the rod and cone layer, but is delayed because the impulse detours through the pigment epithelium. The ERG is advised to distinguish retinitis pigmentosa from similar disseminated choroiditis.

Human perimetry with the ERG remains in the future,[277] since the response is to stray, and not to focused light. Photic driving was said to be best with a blue target of 80 ft-c.[278] Photic driving is more definite in anesthetized or mentally impaired patients. Lesions of the eyes or visual pathways will interfere with photic driving, but ablation of the temporal lobes will improve it. Photic driving will increase abnormal brain waves and facilitate diagnosis. Subjective brightness of the flickering light is greatest at the frequency which drives best, usually at the alpha rhythm.

§ References 256 through 262.

‖ References 264 and 265.

¶ References 268 and 269.

References 270 and 271.

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Correspondence

EFFECTS OF ILLUMINATION AND SLEEP ON ELECTRO-OCULOGRAM

To the Editor:—In his paper entitled "Effects of Illumination and Sleep upon Amplitude of Electro-Oculogram," published in the April, 1955, issue of the ARCHIVES, page 542, Aserinsky refers to a paper entitled "Study of Ocular Movements Under Stress," by K. Lion and R. Brockhurst, which appeared in the ARCHIVES (**46**:315-318, 1951). He states that a plausible explanation for the observed reduction of electro-oculogram (EOG) amplitudes after stress might be that the D. C. potential of the eye was changed by, or during, the experimental procedure, rather than by a decreased excursion of the eyeball, as Lion and Brockhurst suggested. This new explanation is based upon the author's observation that after 5 to 15 minutes of dark adaptation the amplitudes decreased, whereas after light adaptation for 3 to 4 minutes the amplitudes increased. The possibility of a metabolic change affecting the amplitudes of the EOG is suggested.

In the paper by Lion and Brockhurst, it was stated, on page 318, that within 20 to 30 seconds after the stress test was terminated all subjects showed normal ability to fixate accurately. The amplitudes were then of the same magnitude as at the beginning of the stress test. Thus, the increase in amplitude occurred much sooner than the increase which follows light adaptation.

Thus it does not seem that this new information concerning the amplitudes of the EOG is applicable, or contradictory, to the conclusions suggested by Lion and Brockhurst relative to the eye movements after stress.

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THE CINCH OPERATION

To the Editor:—My last paper on the cinch operation appeared in the 1930 *Transactions of the Pacific Coast Oto-Ophthalmological Society*, 25 years ago. Since then I have made a number of changes in its technique. Anyone interested in learning what these changes are should communicate with me, and I will be glad to furnish the desired information.

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Society Transactions

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William E. Krewson 3rd, M.D., *Clerk*

Regular Meeting, Feb. 17, 1955

Ocular Toxoplasmosis: Clinical and Experimental Observations. DR. MICHAEL J. HOGAN, San Francisco.

Aspects of the clinical and laboratory findings in congenital and acquired ocular toxoplasmosis are presented.

Congenital toxoplasmosis is associated with chorioretinitis in 80% of cases. One or both eyes may be affected and single or multiple lesions may develop. The acute inflammation may be present prior to or at birth, or may develop in the neonatal period. Occasional patients develop the initial ocular inflammation any time within the first year after birth. The disease may follow several courses in these patients.

1. Complete quiescence after healing throughout the patient's life.

2. Relapse of a previously healed chorioretinitis as long as 4 to 25 years after the initial disease.

The diagnosis of an acute case in the newborn presents no difficulty. A combination of central nervous system disease, visceral disease, and chorioretinitis, along with a positive methylene blue dye test and complement fixation test, usually is sufficient. Organisms can be isolated from body fluids or tissue in some cases. In late infancy and early childhood the sequelae of the congenital infection appear, namely, hydrocephalus, deviations of the eye, hydrocephalus, and mental retardation.

The methylene blue dye test and complement fixation test are of value in helping to diagnose the disease in the first five years of life. After this time the antibody levels drop and most diagnoses are presumptive. Relapse of the chorioretinitis does not seem to affect the dye-test titer.

In the normal population the incidence of positive dye tests varies from age group to age group (being low in the young and the old and highest in the middle-age groups), in various sections of the country, and in various parts of the world. The general incidence of about 40% of positive dye tests in the general adult population in this country makes the diagnosis of toxoplasmosis of the eye extremely difficult. Unless high titers are present (1:1024 or more), or a fourfold increase or decrease in titer occurs on serial testing, one is not justified in making a presumptive diagnosis. Most uveitis cases have titers of a low level (1:16 to 1:256), and in such cases one cannot place much value on the serologic result. Since the methylene blue dye test is expensive to run, and many aspects of its use and interpretation have not been elucidated, it is not as yet recommended as a routine procedure for the diagnosis of uveitis cases.

The complement fixation test has little value at the present time in the diagnosis of ocular toxoplasmosis.

Skin-testing antigens are of value only for population surveys, and provide little evidence as to the causation in an individual case of uveitis. Most uveitis cases with positive skin tests have dye-test antibodies in the serum in low titer, but the skin test provides no information as to the level of antibody; therefore, it is unsatisfactory for diagnosis. Also, a negative skin test is of no value because a fair percentage of patients have positive dye tests and negative skin tests. Skin-testing antigens also require standardization.

The chorioretinitis of ocular toxoplasmosis is not different from that due to other causes, although most lesions are large, produce heavy vitreous opacification, and tend to recur.

The results of testing 644 patients with various types of uveitis indicate that the incidence of positive dye tests is higher (60%) in the 0 to 20 age group than in the 20 to 50 (30%) and 50 to 70 (20%) age groups. A correlation was obtained between the severity of the chorioretinitis and the presence of a higher dye-test titer.

One patient had bilateral chorioretinitis and a dye-test titer of 1:30,000 nine months after an acute systemic disease.

Patients with chorioretinitis had a much higher incidence of positive dye tests (48%) than those with iridocyclitis (23%). Among a miscellaneous group of 106 patients were 12 with optic neuritis who had low or negative dye tests. All the others, which included cases of scleritis and episcleritis, retinal periphlebitis, Eales's disease, and Coats's disease, showed no serologic evidence of toxoplasmosis.

Aqueous-humor dye tests were found to be of no value in the diagnosis of ocular toxoplasmosis.

A group of patients having suspected ocular toxoplasmosis were treated with pyrimethamine (Daraprim), 25 mg. t. i. d., and sulfadiazine, 1 gm. q. i. d. This treatment, when carried out for three to four weeks, did not seem to affect the inflammatory foci. This result is not surprising, however, when one considers the nature of the parasite and its intracellular localization.

DISCUSSION

DR. HAROLD G. SCHEIE: Dr. Hogan, of what value is the methylene blue dye test in mothers? From a practical standpoint, how far should any practitioner go in studying patients for toxoplasmosis?

DR. MICHAEL J. HOGAN, San Francisco: I think the methylene blue test on mothers is of importance only in helping the person who is investigating toxoplasmosis. If the infant's test is positive, the mother's test almost certainly will be positive, but usually not in as high a titer; so it really is not essential to have a dye test on the mother.

I selected this discussion for tonight because I think we should attempt to survey cases of uveitis as we have always done, employing the means we have at hand. As regards toxoplasmosis, it is very well to perform a skin test, provided the physician keeps his tongue in his cheek and does not place too much emphasis on the result, except in the case of a negative test. I do not think that one should request laboratories or the public health service to perform routine methylene blue dye tests on patients with uveitis, for the reason that the result of a single test is of no value and it is impossible at present for any laboratory to carry out serial tests on patients with chorioretinitis. Therefore, I should be content with ruling out all other possible causes of uveitis; and if the results were negative, and if the patient had findings of what I thought were possible chorioretinal lesions of toxoplasmosis, I would consider treatment with pyrimethamine and sulfadiazine and would watch the lesion very closely. It is possible to give a smaller amount of pyrimethamine in a dose of 25 mg. twice a week plus sulfadiazine, 2 gm. a day, over a 15- to 30-day period. This regimen might prevent the escape of organisms from cells and their proliferation in the extracellular tissues, and therefore favor gradual healing of the disease. I think that would be the next step in our investigations. It would be much safer from the standpoint of the patient, too; but at present I do not think we should encourage routine testing of sera. Our experience, as presented here, demonstrates the inadequacies in the diagnosis of this disease.

DR. W. O. LAMOTTE: Is there any evidence that the performance of skin tests affects in any way the positivity of a subsequently performed methylene blue dye test?

DR. MICHAEL J. HOGAN, San Francisco: There is a variation of opinion on this point. Some observers feel that the skin test results in positive dye tests, and others feel that the opposite is so. Therefore it is all right to skin-test if you wish a dye test on a patient. If one does a skin test, and then takes serum any time within the subsequent 48-hour period, the test will not affect the serum antibody level. After the 48-hour period, when the skin test becomes positive, the serum antibody may rise. This point needs further clarification.

DR. IRVING H. LEOPOLD: Have the titers been correlated among the different laboratories, and is there any practical significance of serum neutralization? Is the neutralization test used at all any longer?

DR. MICHAEL J. HOGAN, San Francisco: The rabbit neutralization test is not done any more, so far as I know. We gave it up quite a while ago because we

found that we could produce a positive test, depending on the part of the rabbit's back in which we injected the antigen. There is fairly good correlation among various laboratories, and we have checked our results with several groups. There is not too much difference in the outcome of the results of most of the testing that has been done. We have found that our results are reproducible on the same sera. Our results with experimental infections have also been satisfactory.

QUESTION FROM THE CHAIR: Can the average clinical laboratory do this test?

DR. MICHAEL J. HOGAN, San Francisco: No, the test is rather complicated. It is not an easy test to do and is fraught with some danger. One has to handle live organisms, and Toxoplasma is one which is not safe to be careless with. Four laboratory technicians whom I know of have acquired acute toxoplasmosis after the handling of organisms. Some deaths have occurred.

The test also is fairly expensive, and the average laboratory would not find it practical to run a few tests. Therefore I think it not a test which can be run by the average laboratory. If some of you feel that you wish to have it done, it might be advisable to get your state public health laboratory to set up tests for your own particular area. We have been willing to test sera for physicians who send them in suspected cases, and we get a fair number of sera from patients with degenerative diseases. We have been testing sera sent by mail and have found it impractical; so we try to discourage the testing of sera except on patients with active and progressive chorioretinitis.

DR. ARTHUR H. KEENEY: How early in life can calcifications be found in toxoplasmosis, and do calcifications ever occur in adult toxoplasmosis affecting a laboratory worker?

DR. MICHAEL J. HOGAN, San Francisco: One laboratory worker who died had toxoplasmosis with involvement of the viscera, brain, lungs, liver, and spleen. The disease progressed so rapidly that the patient died before degenerative changes occurred in the brain. Calcifications in children can develop fairly quickly—I should say in one to two months. In some of the children who develop the disease prior to birth the calcifications are already present at birth. In those children who developed the disease one month after birth, a calcification usually can be found at the second to the third month. Calcification is not an important sign of adult acquired toxoplasmosis.

DR. E. H. HEYDT, Jenkintown, Pa.: Do any special precautions have to be taken in the collection of blood specimens and in their shipment to the laboratories for examination?

DR. MICHAEL J. HOGAN, San Francisco: Whole blood is collected and allowed to clot. The separated serum can be placed in a Wassermann tube and sealed by flame. It can then be shipped by ordinary mail or by airmail, and does not have to be registered. We usually add a little thimerosal (Merthiolate) when we ship, but if the serum is handled carefully and put in sterile Wassermann tubes, there is very little danger of contamination.

QUESTION: When one is confronted with a case of toxoplasmosis, can one usually obtain a history of any systemic disease compatible with toxoplasmosis which might have been acquired in adult life?

DR. MICHAEL J. HOGAN, San Francisco: Usually not. It is unfortunate that so far no one has been able to uncover any transmitting agent which is common in all mothers who have given toxoplasmosis to children. A few mothers have been exposed to cats or dogs, and the dogs were said to have sickened and died during the course of pregnancy. We know the disease can exist in dogs, and it is possible that in some way infected household animals or nearby pets may make it possible for the disease to be transmitted to the affected mother. The mother, strangely, never shows evidence of the illness during the course of her pregnancy.

DR. WILLIAM T. HUNT: Has toxoplasmosis ever been found in a child?

DR. MICHAEL J. HOGAN, San Francisco: In only one case that I know of. Dr. Arlington Krause, of Chicago, described it to me personally. The mother gave birth to two consecutive children with toxoplasmosis.

Obituaries

ROBERT RHODES CHACE, M.D.
1910-1954

On Nov. 24, 1954, Dr. Robert Rhodes Chace died suddenly at his home in Bronxville, N. Y., at the age of 44. He was born in Providence, R. I., where he attended the Moses Brown School and Brown University. He was elected to Phi Peta Kappa and graduated in 1933. His medical education was at Yale School of Medicine, and his early interest in ophthalmology was shown by his publishing a paper upon "The Grafted Eye and the Regeneration of the Lens in Amphibians" while an undergraduate and his selection of a similar subject for his thesis for his medical degree.

After his graduation from medical school, in 1937, he served an internship at the Rhode Island Hospital, then a residency at the Institute of Ophthalmology in New York, and he received the degree of Doctor of Medical Science from Columbia University in 1943. He joined the staff of the Institute of Ophthalmology as Assistant Ophthalmologist and Instructor in Ophthalmology of Columbia University College of Physicians and Surgeons. He was also on the staff of the Lawrence Hospital, Bronxville. He was the author of articles on physiological optics, clinical ophthalmology, and the testing of color vision and for malingering.

He was consultant to the division of vocational rehabilitation of the New York State Department of Education. Dr. Chace enjoyed teaching and was held in high regard by those who had attended his lectures.

He was certified by the American Board of Ophthalmology and a Fellow of the American College of Surgeons, the New York Academy of Medicine, and the American Academy of Ophthalmology and Otolaryngology. He was a member of the Association for Research in Ophthalmology, the Westchester County Medical Society, the Medical Society of the State of New York, and the American Medical Association.

JOHN P. MACNIE
J. VINCENT FLACK

News and Comment

GENERAL NEWS

National Council to Combat Blindness, Inc.—The National Council to Combat Blindness, Inc., has added to its research program the financing of summer fellowships initiated by its Scientific Advisory Committee as a stimulus to the development of ophthalmic investigators. All applicants for fellowships are required to make their own arrangements for suitable research facilities with accredited institutions.

The National Council to Combat Blindness Grants-in-Aid and Fellowship awards for the fiscal year 1954-1955, as approved by its Scientific Advisory Committee are:

Retina Foundation, Boston; Endre A. Balazs, M.D. ($4,000): Synthesis of Hyaluronic Acid in the Vitreous Body of Embryos and Young Animals.

University Hospitals, State University of Iowa; Hermann M. Burian, M.D. ($2,000): Temporal Relationships of Electric Responses and Chronaxie of Human Retina (Continuation)

Ohio State University, College of Veterinary Medicine; Clarence R. Cole, D.V.M. ($2,000): Ocular Toxoplasmosis in Domestic Animals

University of Buffalo Medical School; Angelos N. Dellaporta, M.D. ($2,000): Pathological Studies on Experimental Retinal Detachment

Ohio State University, Department of Optometry; Vincent J. Ellerbrock, Ph.D. ($960): Compilation of a Volume on Research in Subnormal Vision, Including Evaluation of All Aids

The Presbyterian Hospital Medical Center, Institute of Ophthalmology, New York; William G. Everett, M.D. ($250): Mensuration of the Human Eye by X-Ray and the Relation of Measurements to Pathologic States (Continuation)

New York Eye and Ear Infirmary; Bernard Goldberg, M.D.; Ralph Levene, M.D.; Gerald Kara, M.D. ($1,000): The Possible Role of Hyperestrinism in the Pathogenesis of Retrolental Fibroplasia (Continuation)

Wills Eye Hospital, Philadelphia; Harry Green, Ph.D.; Irving H. Leopold, M.D. ($3,000): Lens Metabolism and Cataract Formation (Continuation)

New York Eye and Ear Infirmary; Jerry Hart Jacobson, M.D. ($2,400): Electroretinography in Retinal Diseases

University of California Medical Center, San Francisco; Samuel J. Kimura, M.D. ($1,200): Study of Keratitis Sicca and Sjögren's Syndrome

University Hospitals, State University of Iowa; P. J. Leinfelder, M.D. ($1,500): Effect of Change in pH in the Aqueous on Metabolism of the Lens and Cornea

Government Hospital, Haifa, Israel; I. C. Michaelson, M.D. ($2,800): New Vessel Growth in the Eye, Particularly in the Retina and Cornea (Continuation)

Kumamoto University Medical School, Japan; Yukihiko Mitsui, M.D. ($2,650): Cultivation of Trachoma Virus in Cultivated Human Conjunctival Epithelium by Roller Tube

Ziya Gün Institute for Research in Trachoma, University of Istanbul, Turkey; F. Necdet Sezer, M.D. ($2,000): Cultivation of Trachoma Virus (Continuation)

Institute of Ophthalmology, Columbia University; George K. Smelzer, Ph.D. ($1,200): Investigation of Experimentally Produced Exophthalmos

Royal College of Surgeons and the Royal Eye Hospital, London; Arnold Sorsby, M.D. ($3,000): The Biochemistry of Hereditary Retinal Lesions

Manhattan Eye, Ear and Throat Hospital, New York; C. C. Teng, M.D. ($2,500): Optic Nerve Study

College of Physicians and Surgeons, Columbia University; Ludwig von Sallmann, M.D. ($1,000): Clinical and Experimental Studies in the Distribution of P^{32} in Tumors of the Eye

Albert Einstein Medical Center, Philadelphia; Charles Weiss, M.D., Ph.D. ($200): Exhibit on "Laboratory Aids in the Diagnosis of Infections of the Eye" (Bacteriologic, Serologic, Myocologic, Virologic, and Parasitologic Procedures), shown at the International Congress of Clinical Pathologists, Washington, D. C., Sept. 6-11, 1954

Eye and Ear Hospital, University of Pittsburgh Medical Center; Jerome J. Wolken, Ph.D. ($3,000): Photoreceptor Structures

A National Council to Combat Blindness full-time research fellowship has been awarded to Robert K. Abraham, M.D. Dr. Abraham is a graduate of the University of Illinois College of Medicine, spent three months at Johns Hopkins, under Frank B. Walsh, M.D., and completed one year as a Fellow-in-Ophthalmology at the Kresge Eye Institute. He is a resident of Wayne University Graduate School of Medicine. As a National Council Fellow, Dr. Abraham will spend six months at the Institute of Ophthalmology, London, under the supervision of Sir Stewart Duke-Elder and Norman Ashton, M.R.C.S., in a project study concerned with the research techniques in glaucoma, the remaining six months to be spent at an accredited institution in the U. S.

The Council's Scientific Advisory Committee has been enlarged to 16 members, and now includes:

James H. Allen, M.D., Chief
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New York Hospital, Cornell Medical Center
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University of California School of Medicine
San Francisco

Irving H. Leopold, M.D.
Director of Research & Attending Surgeon
Wills Eye Hospital
1601 Spring Garden St.
Philadelphia

ANNOUNCEMENTS

Appointment of Dr. Ludwig von Sallmann to National Institute of Neurological Diseases and Blindness.—Dr. Ludwig von Sallmann, Professor of Ophthalmology at Columbia University College of Physicians and Surgeons, and Attending Ophthalmologist at the New York Presbyterian Hospital, will join the National Institute of Neurological Diseases and Blindness, Public Health Service, Bethesda, Md., as Visiting Scientist, on Aug. 1, 1955, in order to direct and expand its eye research program at the Clinical Center. Dr. von Sallmann will continue his studies there on ocular neurophysiology and ocular pharmacology and is now actively recruiting a research staff for these and other investigations.

Executive Committee of the Section on Ophthalmology, American Medical Association.—The Executive Committee of the Section on Ophthalmology is calling a meeting to discuss certain phases of ophthalmological practice, to be held in the St. Dennis Room of the Dennis Hotel in Atlantic City at 8:00 p. m., June 7, 1955. All ophthalmologists are invited to attend.

HAROLD G. SCHEIE, M.D.
Secretary
Section on Ophthalmology

Books

The Study of the Brain. By Hyman S. Rubinstein. Price, $9.50. Pp. 209, with 93 illustrations and 44 plates. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1953.

One of the chief difficulties in teaching neuro-ophthalmology to residents is their paucity of knowledge of the anatomy of the brain and spinal cord. Topical diagnosis depends upon a complete familiarity with nerve pathways subserving particular functions; for example, the detection of an internuclear paralysis pinpoints the lesion to a space of approximately 0.5 cm. in the brain stem. It is urgent, therefore, that all those who expect to familiarize themselves with neuro-ophthalmology shall be well founded in neuroanatomy, or at least those parts which apply particularly to the eye. In 1947 Rubenstein and Davis published an excellent stereoscopic atlas of neuroanatomy (New York, Grune & Stratton, Inc.). The present text is a companion text to this stereoscopic atlas, but is complete in itself, since the key drawings of the "Atlas" have been included.

Designed primarily for the student beginning his study of neuroanatomy, Chapter I is a dissecting manual, outlining the steps necessary to expose the various desired details. The rest of the text deals with the structure of the cerebrum and cord, largely from the point of view of function. It is an excellent text for anyone desirous of reviewing his neuroanatomy, and should certainly be included with any well-stocked ophthalmological library.

Atlas—Diseases of the Eye. Section I. Prepared by the Medical Illustrations Department, Institute of Ophthalmology, in association with Moorfields, Westminster & Central Eye Hospital, London. Roche Products, Ltd., 1955.

This "Atlas," published by Roche Products, Ltd., a pharmaceutical firm in England, is to appear in six sections. Section I presents the fundus in systemic diseases. This volume consists of 32 beautiful fundus drawings, all but one in color. The subjects illustrated are the normal fundus, the changes in arteriosclerosis and hypertension, diabetic retinopathy, arterial and venous occlusions, retinal perivasculitis, the retinopathy of pregnancy, renal retinopathy, the retina in pernicious anemia, and leukemic changes in the retina. This is a valuable atlas for teaching purposes. These "Atlases" are distributed without charge, and probably can be obtained by contacting the Roche Products representative in the United States.

Medical Progress—1955. Edited by Morris Fishbein. Price, $5.00. Pp. 346. McGraw-Hill Book Company, Inc., 330 W. 42d St., New York 36, 1955.

This volume is the third in a series which began in 1953. Each chapter covers a division of medicine or surgery. The chapter on ophthalmology was prepared by Dr. Manuel L. Stillerman, and consists of nine pages, dealing with retrolental fibroplasia, infantile glaucoma, the use of acetazoleamide (Diamox) in glaucoma, and radioctive phosphorus in the detection of intraocular cancer. Although Dr. Stillerman has prepared his summaries well, they are so restricted in scope that they are of little benefit to an ophthalmologist, and it is a good guess that an internist or a general surgeon would gain little useful information in these subjects by a perusal of these nine pages. Similarly, it is hardly to be expected that an ophthalmologist would learn very much from the other nineteen chapters. The material contained in this book can be better culled from the journals as they come off the press. The phrase "too little and too late" seems to apply here. It is difficult to see the usefulness of a book of this character.

Advanced Surgery of Cataract. By Daniel B. Kirby, M.D. Price, $27.00. Pp. 271, including index, with 138 figures, 96 illustrations, and 22 color plates. J. B. Lippincott Company, E. Washington Sq., Philadelphia 5, 1955.

As explained in the foreword, this book was completed on Sunday, Dec. 27, 1953, the day on which Dr. Daniel B. Kirby succumbed to a heart attack. In his first book, "The Surgery of Cataract," published in 1950, there were many controversial issues upon which Dr. Kirby did not take a definitive position, probably feeling that in a book of that character all sides should be presented without bias. In this book Dr. Kirby has stated his position, and the main framework of the book is built of his own ideas, which those who knew him well realized were frequently original and decided—for example, the use of curare in cataract surgery. Like the first, this book is beautifully illustrated, and the two volumes together constitute a monumental work. Ophthalmology is deeply indebted to Kirby for this complete documentation of our knowledge of cataract surgery.

Embryology of the Human Eye. By Aleta Nichols Barber. Price, $8.75. Pp. 236, including index and glossary, with 193 illustrations. C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1955.

Dr. Barber has had wide experience in the preparation of this book, which represents her notes and material in teaching embryology of the eye for over a decade. As Dr. Holman states in his foreword, "This book is neither a textbook nor an atlas, but some of both. While it is designed primarily for students and practitioners of ophthalmology, other biologists, especially embryologists, anatomists and pathologists, will find it a valuable source for material." The illustrations are beautiful, and the material is well organized into four main sections: (1) embryogenesis, (2) organogenesis, (3) differentiation, and (4) age-length correlation. The last is especially valuable, since chronologic and linear measurements vary in different embryos.

Angiographic Localization of Intracranial Masses. By Ecker and Paul A. Reimenschneider. Price, $13.50. Pp. 433, with 413 illustrations. Charles C Thomas, Publisher, 301 E. Lawrence Ave., Springfield, Ill., 1955.

The authors published the details of the technique used in angiography, and their concept of normal films, in an earlier publication, entitled "The Normal Cerebral Angiogram," published by Charles C Thomas in 1951. The present volume is a reference atlas, consisting of a series of cerebral angiograms in proved cases of intracranial masses. The authors stress the fact that the films reproduced have not been retouched and are from proved cases—almost all by microscopic examination of tissue removed either at operation or at necropsy.

The book is divided into two parts, the first of which is a general description of the material. From an angiographic point of view, the authors consider twenty-seven specific regions in which intracranial masses occur. The second part of the book is devoted to a chapter on each of these regions. The book is actually a manual which will be much more useful for radiologists, neurologists, neurosurgeons, and neuropathologists than for ophthalmologists. It undoubtedly is an outstanding contribution to the techniques of this field.

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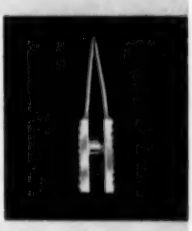

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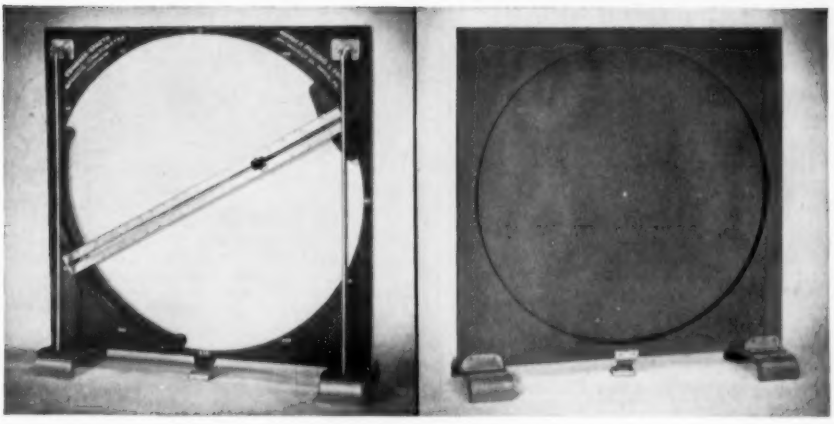

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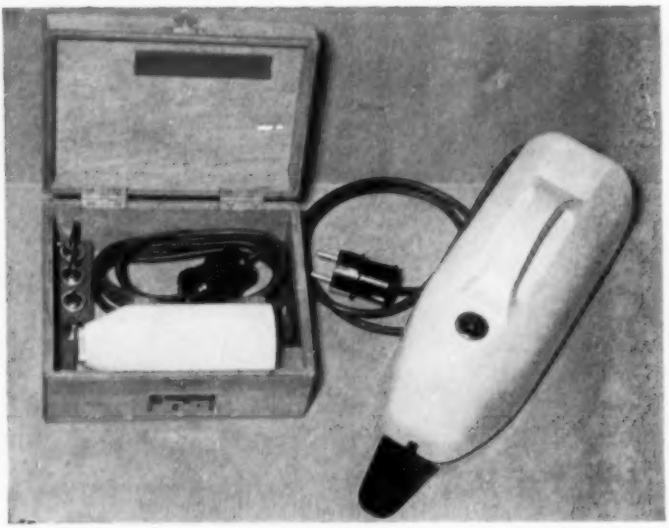

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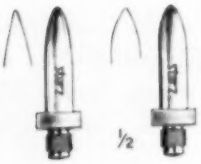

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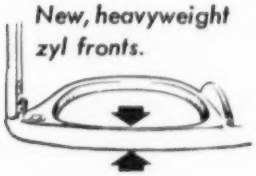

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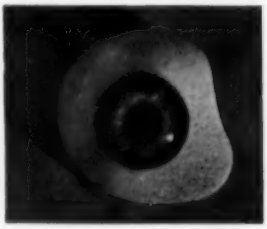

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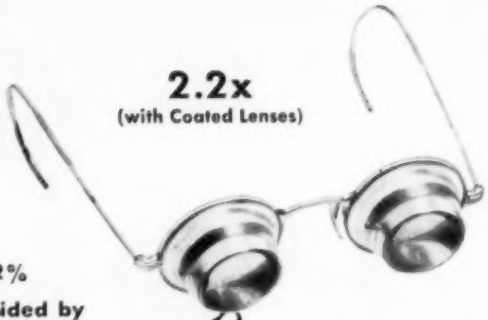

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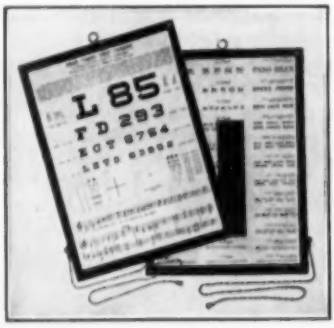

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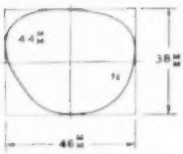

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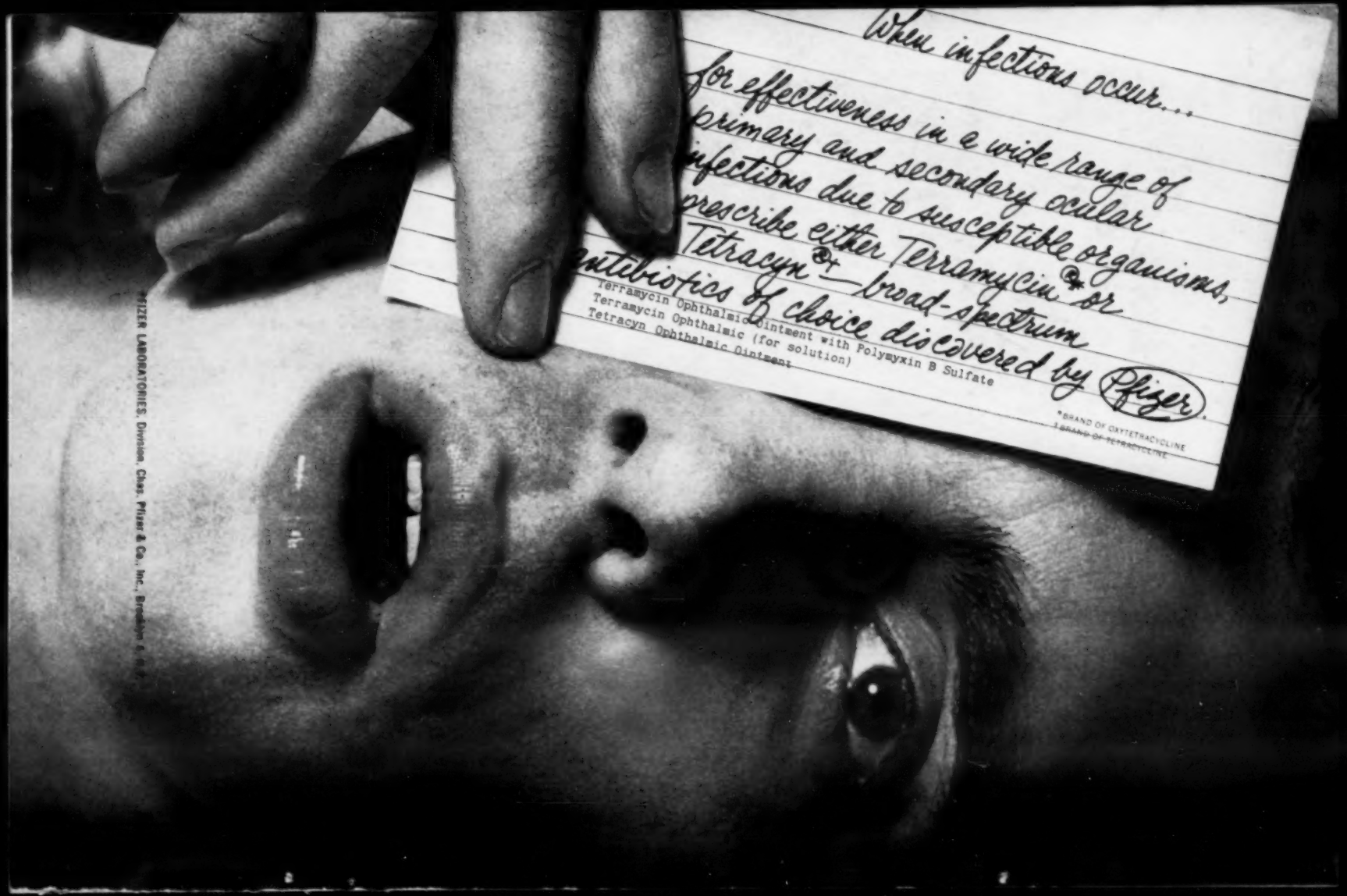
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